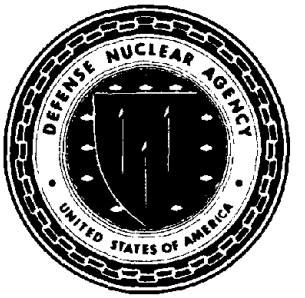


Defense Nuclear Agency
Alexandria, VA 22310-3398

DNA-TR-90-157

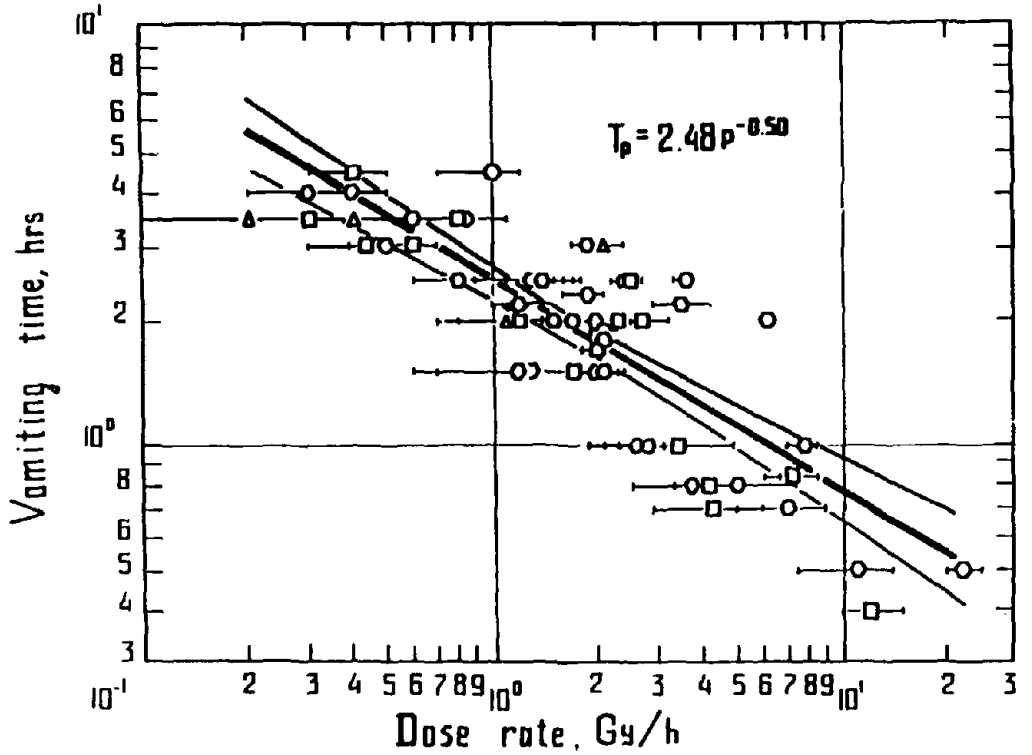
Biological Effects of Protracted Exposure to Ionizing Radiation: Review, Analysis, and Model Development

George H. Anno, et al.
Pacific-Sierra Research Corporation
12340 Santa Monica Boulevard
Los Angeles, CA 90025-2587

November 1991

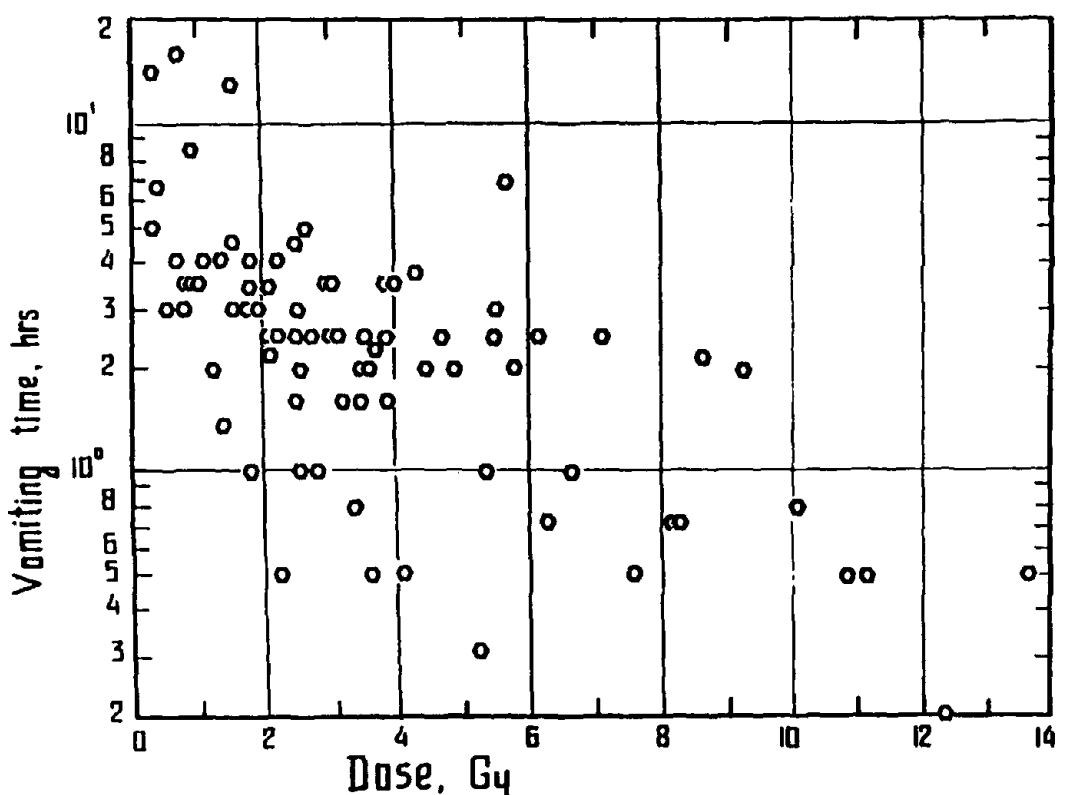
Technical Report

CONTRACT Nos. DNA 001-86-C-0307
and DNA 001-87-C-0104



Source: Baranov and Guskova [1988].

Figure 3. Vomiting time versus dose rate.



Source: Baranov and Guskova [1988].

Figure 4. Vomiting time versus dose.

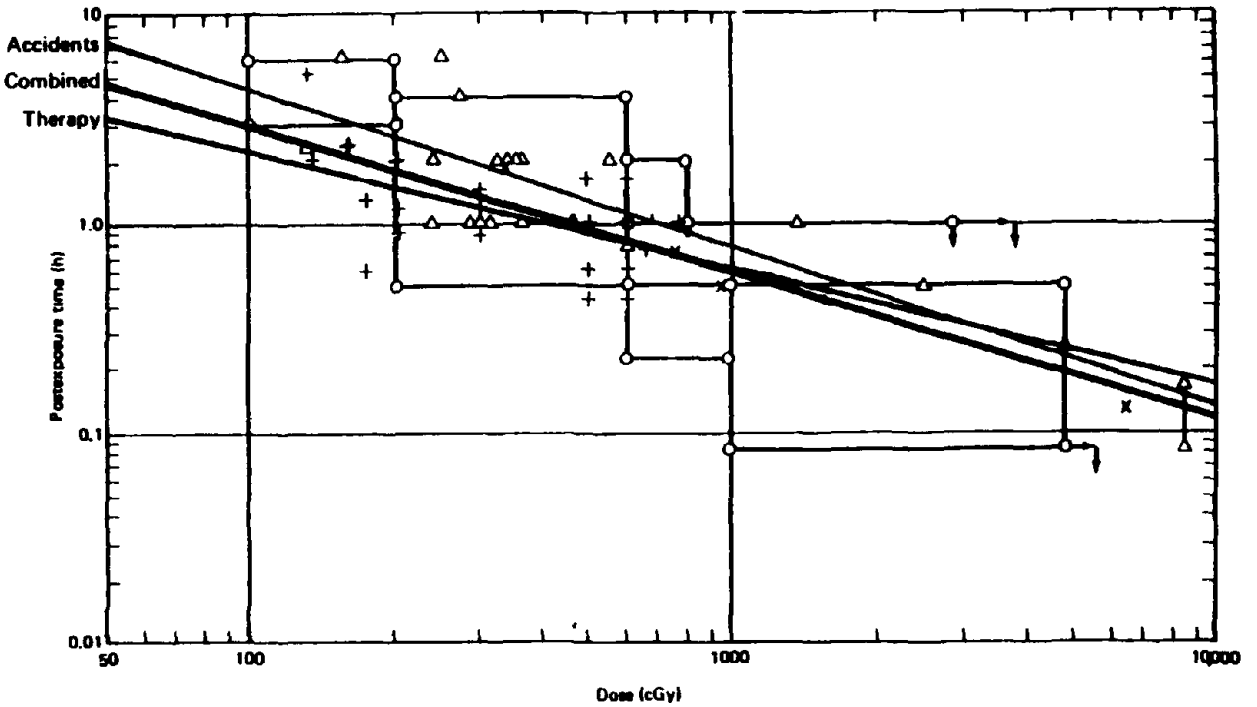


Figure 5. Onset of prodromal symptoms related to dose.*

*

△	Accident	Brucer 1959; Cronkite et al. 1956; Fanger and Lushbaugh 1967; Hübner and Fry 1980; Karas and Stanbury 1965; Laumets 1965; Lushbaugh 1973; Thoma and Wald 1959; Wald and Thoma 1961.
+	Therapy	Court Brown 1953; Miller et al. 1958; Rider and Hasselback 1968; Salazar et al. 1978.
O	Composite	Gerstner 1958; Gerstner 1970; Glasstone and Dolan 1977; Laumets 1965; Lushbaugh 1973; NATO 1973; Warren and Grahn 1973; Zellmer 1961.
X	Expert Opinion	Anno 1983; Fanger and Lushbaugh 1967; Gerstner 1958; Glasstone and Dolan 1977; Saenger 1963; Thoma and Wald 1959; Wald and Thoma 1961.

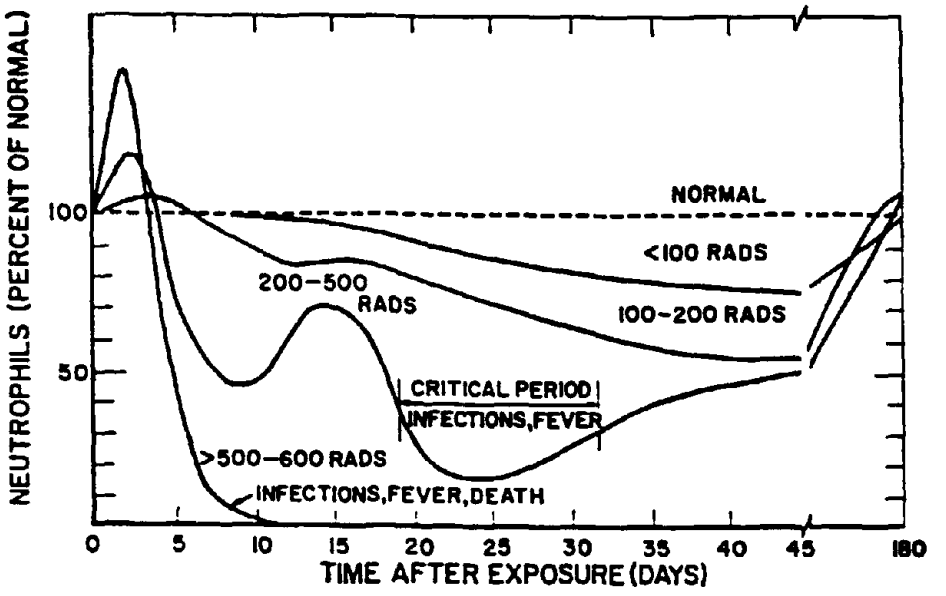
100 cGy/h. Vomiting onset time (h), referenced from the end of exposure, was found to be related to dose (cGy) given by,

$$T = 10^{\alpha} \cdot D^{\beta}$$

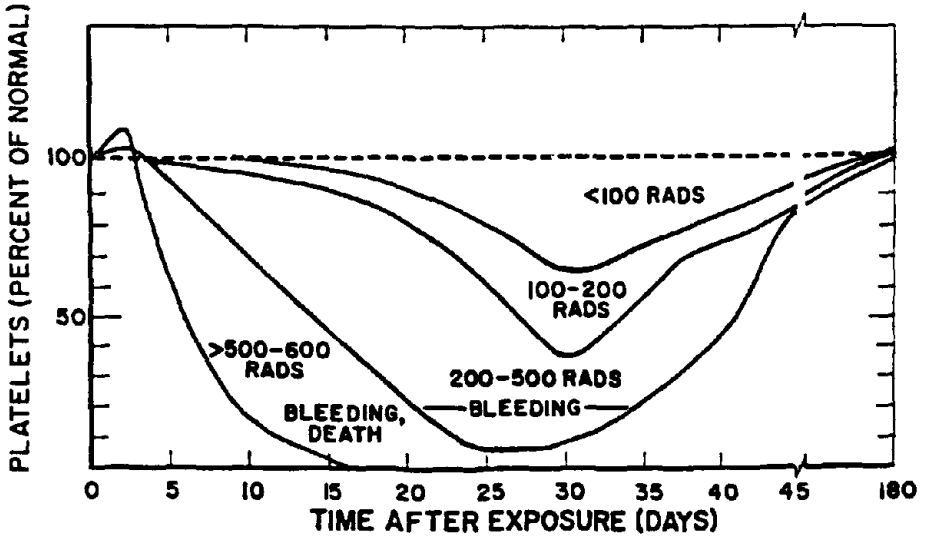
where

Data Category	α	β
Accidents (23 pts.)	2.1830	-0.77260
Therapy (25 pts.)	1.4611	-0.56271
Combined (52 pts.)	1.8641	-0.69022

Neutrophils

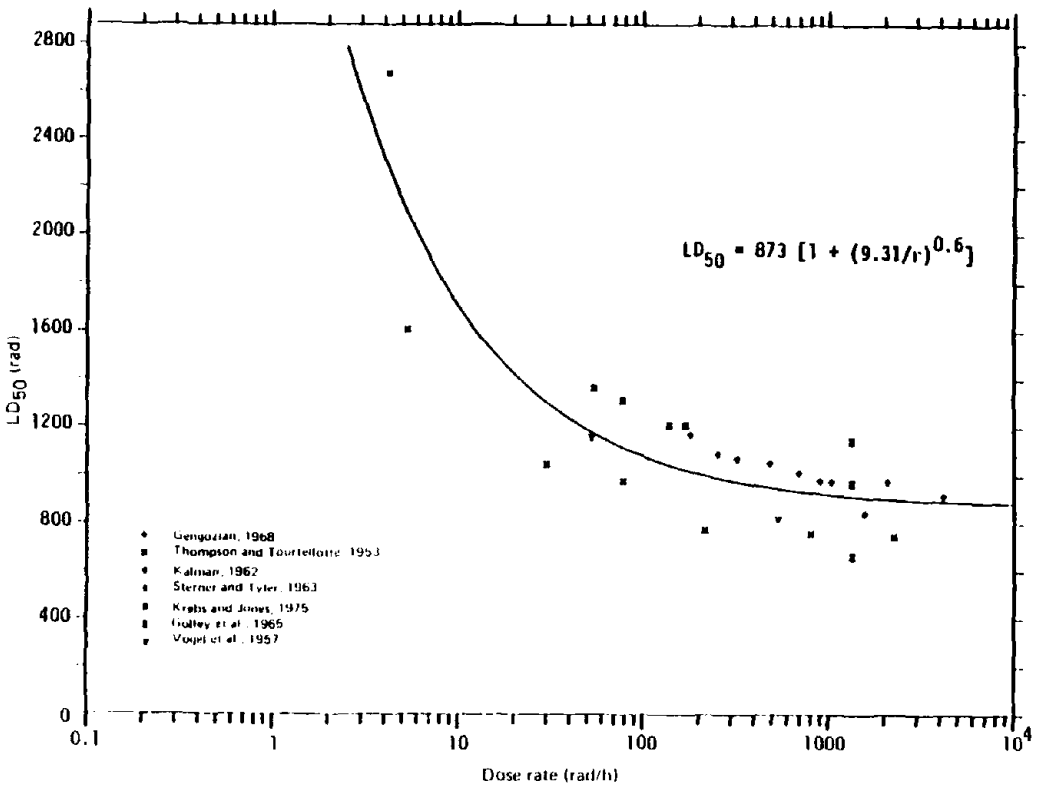


Platelets



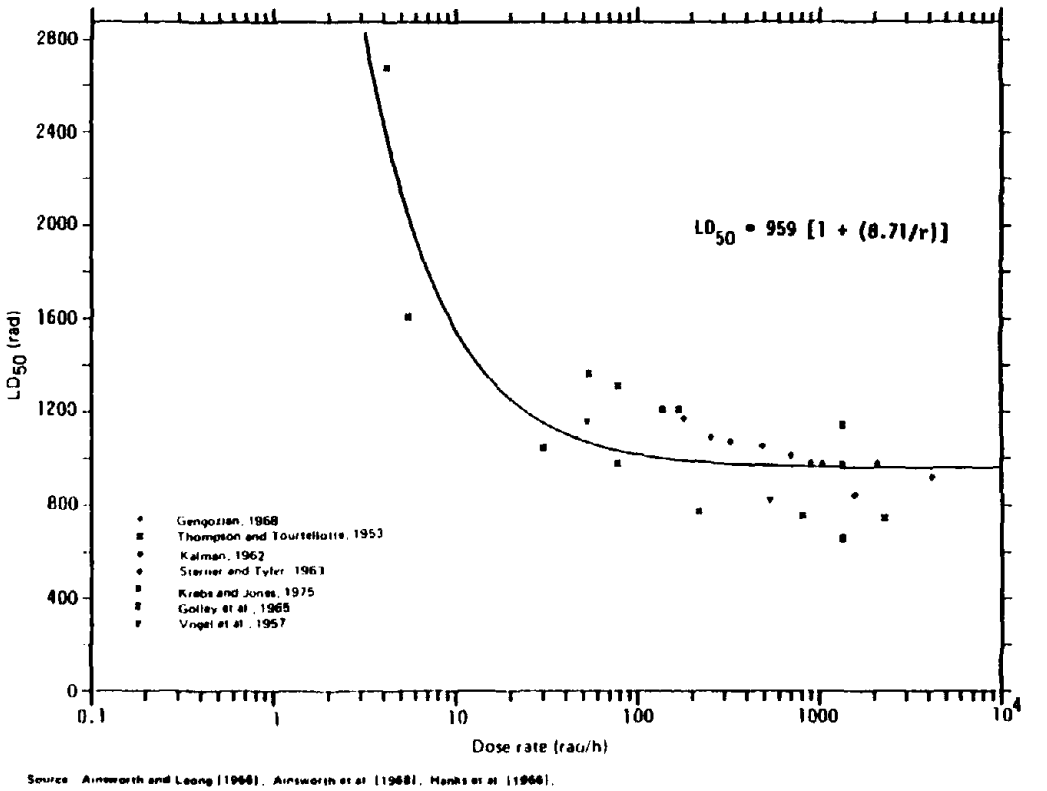
Source: Langham (1967)

Figure 22. Blood count levels following single acute radiation exposures.



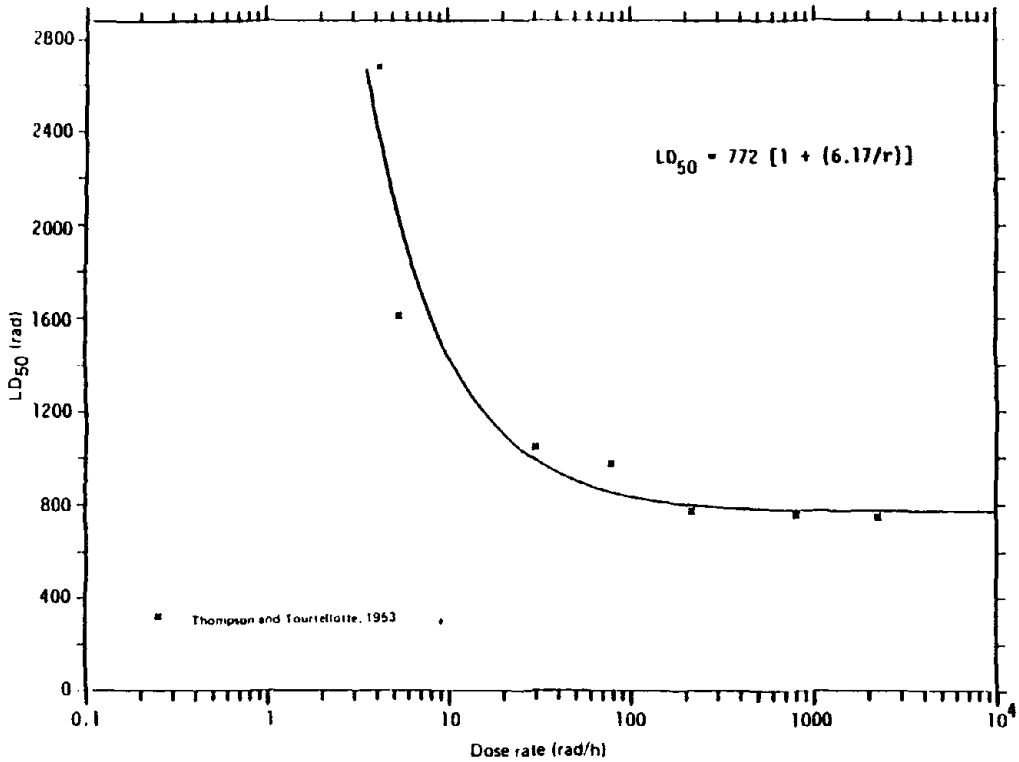
Source: Ainsworth and Leong (1966), Ainsworth et al. (1968), Hanks et al. (1966), Nachtway, Ainsworth, and Leong (1967), Page (1968), Page et al. (1965).

Figure 27. LD_{50} versus dose rate--three parameter model for mouse.



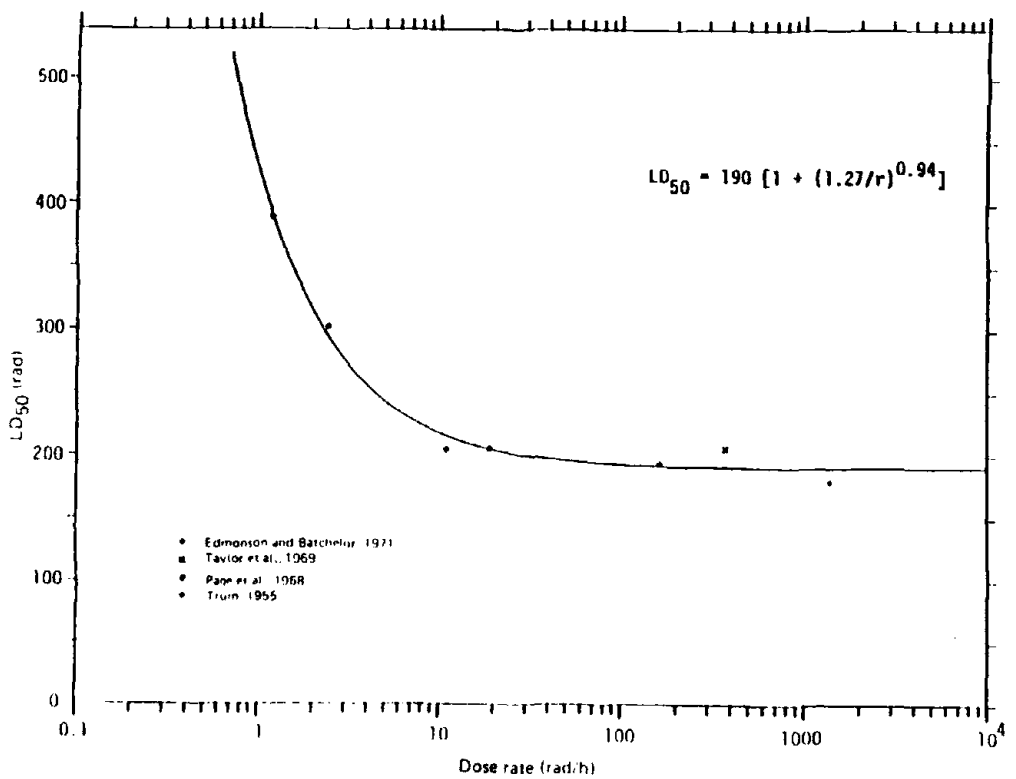
Source: Ainsworth and Leong (1966), Ainsworth et al. (1968), Hanks et al. (1966), Nachtway, Ainsworth, and Leong (1967), Page (1968), Page et al. (1965).

Figure 28. LD_{50} versus dose rate--two parameter model for mouse.



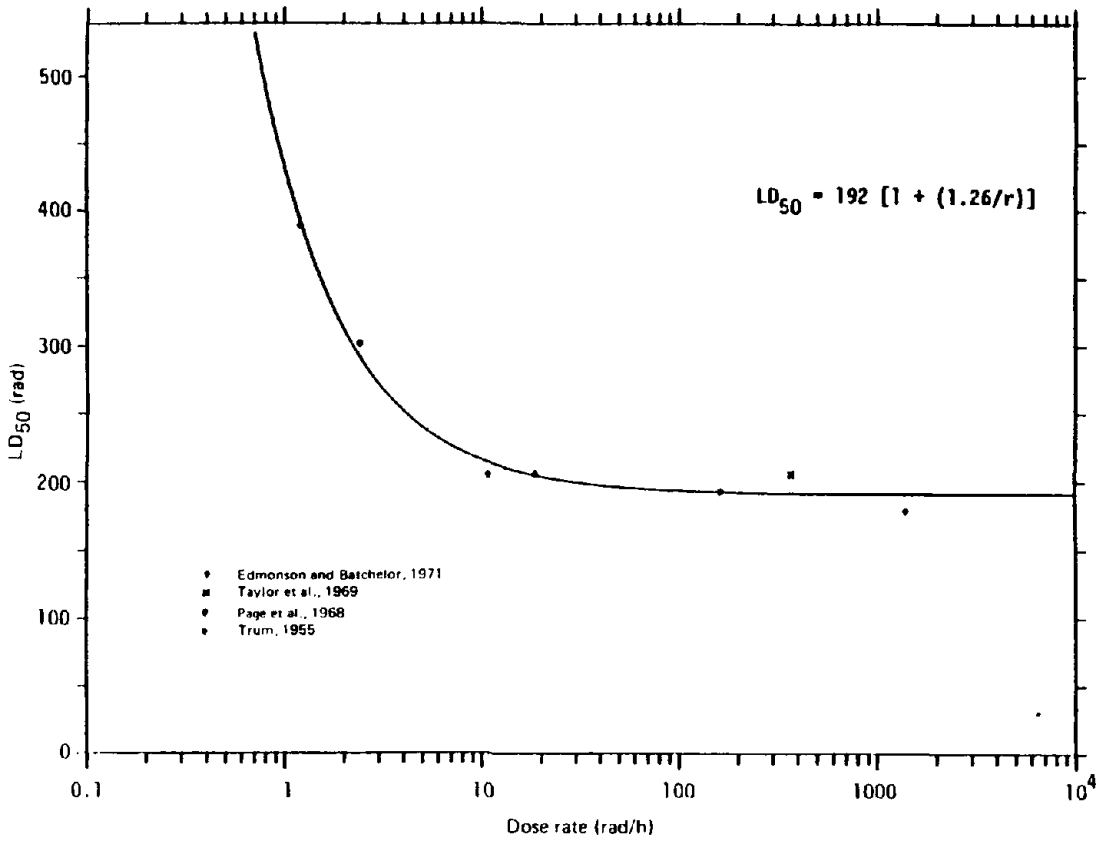
Source: Ainsworth and Leong (1966); Ainsworth et al. (1968); Hanks et al. (1966); Nachtway, Ainsworth, and Leong (1967); Page (1968); Page et al. (1965).

Figure 29. LD_{50} versus dose rate--two parameter model for mouse, from Thompson and Tourtellote (1953) only.



Source: Ainsworth and Leong (1966); Ainsworth et al. (1968); Hanks et al. (1966); Nachtway, Ainsworth, and Leong (1967); Page (1968); Page et al. (1965).

Figure 30. LD_{50} versus dose rate--three parameter model for sheep.



Source: Ainsworth and Leong [1966]; Ainsworth et al. [1968]; Hanks et al. [1966]; Nachtway, Ainsworth, and Leong [1967]. Page [1968]; Page et al. [1965].

Figure 31. LD_{50} versus dose rate--two parameter model for sheep.

An animal radiation study by Soviet researchers Grigorev, Gorlov, and Shafirkin [1978] was translated into English and reviewed. In that study, the effects on LD_{50} of chronic (constant) dose rate exposure, ranging from 1 to 932 rads/day, was reported for a variety of animal species, including mice, rats, guinea pigs, rabbits, donkeys, sheep, goats, dogs, and monkeys. Empirical modeling was performed that included an extrapolation to man based on a polynomial relationship linking basal and water metabolism, animal weight, period of semirecovery from radiation damage, period of maximum white blood cell (WBC) depression, and lifetime of erythrocytes. Biological effectiveness was defined in terms of a dose-rate dependent coefficient that is the ratio of the effective dose D_o (i.e., acute LD_{50}) to the accumulated dose, D, given as,

Table 19. Parameters for LD_{50} versus dose rate.

Species	s	R	D_o	dgf	$(RMS/dgf)^{1/2}$	Figure No.
Swine	0.9394 ± 0.0321	21.74	229	14	32.51	9
Swine	1.0	18.77	235	15	32.32	10
Goat	1.0206 ± 0.2160	2.089	240	4	58.94	9
Goat	1.0	2.146	239	5	52.76	10
Dog	0.7805 ± 0.0913	5.482	263	14	31.78	9
Dog	1.0	4.074	270	15	33.12	10
Sheep	0.6887 ± 0.1931	1.960	162	8	23.33	9
Sheep	1.0	1.614	170	9	24.29	10
Sheep[a]	0.9423 ± 0.1472	1.272	190	4	11.21	14
Sheep[a]	1.0	1.258	192	5	10.21	15
Mice	0.5995 ± 0.0856	9.307	873	24	202.1	11
Mice	1.0	8.717	959	25	202.4	12
Mice[b]	1.0	6.173	772	4	236.0	13

[a]Sheep--without data from Hanks et al. [1966].
[b]Mice--data from Thompson and Tourtellotte [1953] only.

$$\frac{D}{D_o} = \left[1 - b \log_{10}\left[\frac{720}{r}\right]\right]^{-1} ,$$

where r is the dose rate in rads per hour; b = 0.29 for small animals (mice, rats, and guinea pigs); and b = 0.22 for large animals and humans. When the dose rate, r, is less than or equal to the approximate values listed below, D/D_o can be expressed by the simple relationship,

$$D/D_o = 1/ar ,$$

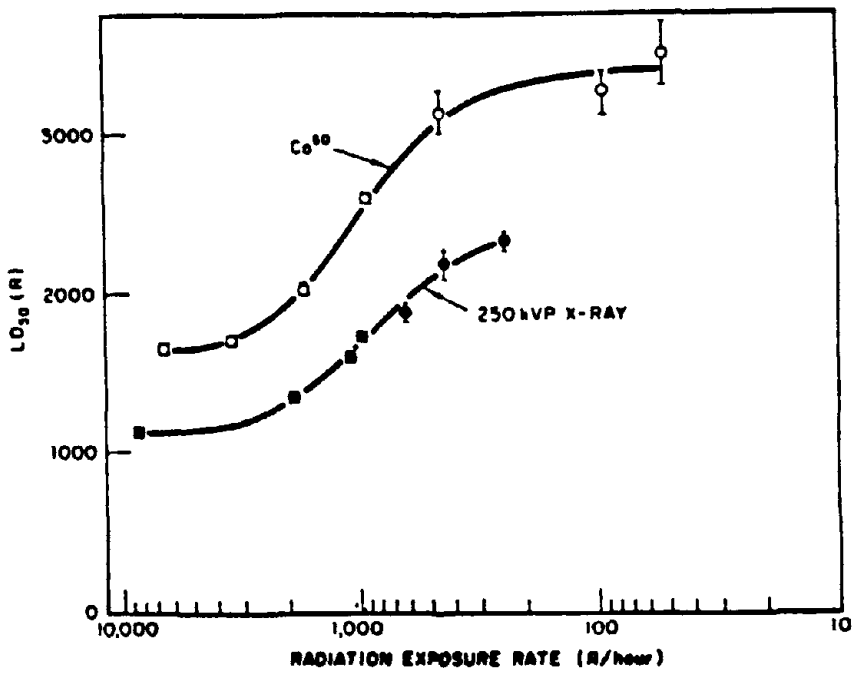
where:

	a(h/rad)	r(rad/h)
Mice	0.055	8.3
Rats	0.084	4.6
Large animals	0.168	2.5
Monkeys	0.36	1.3
Man	0.48	0.63

Although our review of the dose-rate dependent relationships above does not include comparisons with other empirical models, such as the form for LD_{50} used to fit the animal data shown in Figs. 9 through 15, and those discussed below (Constant Dose Rate Models). Grigorev, Gorlov, and Shafirkin [1978] point out that D/D_o does not depend on the dose rate beyond 720 rads/h, as can be seen in the first equation above.

2.4.2 Gastrointestinal Damage.

Using the split dose technique, Krebs and Leong [1970] performed a study with mice to determine the effect of constant exposure rates on the gastrointestinal $LD_{50/5}$ for both ^{60}Co and 250 kVp X-ray irradiation. A plot of their results are given in Fig. 32 that indicates a factor of about two increase in $LD_{50/5}$ when the dose rate decreases from about 8400 to 240 R/h for the 250 kVp X-rays and from about 6700 to 52 R/h for ^{60}Co gamma rays. The curves, based on probit regression fit of mortality, are separated by a factor of 1.48 which represents the effective RBE between the two types of photon radiation over the range of exposure rate. It should be mentioned that the two lowest dose rate points for ^{60}Co irradiation 52 and 93 R/h were developed from actual exposure periods of only 18 h plus a required "topping dose" given at a high dose rate (8400 R/h) to extend the radiation to lethality. Based on this data, however, the corresponding inferred exposure periods would be about 35 and 67 h. It is possible that the $LD_{50/5}$ values would have been significantly higher at those two low dose rates had the exposure periods been allowed to full-term lethality. If so, the exposure periods would have been in excess of those estimated from their actual 18-h exposures.



Source: Krebs and Leong [1970].

Figure 32. Effect of dose rate on gastrointestinal $LD_{50/5}$ in mice.

All actual exposure periods in the Krebs and Leong work were well short enough to avoid proliferation of the intestinal epithelium to ensure that the $LD_{50/5}$ increase with decreasing dose rates only involved Elkind-type repair. Accordingly, an injury repair model was constructed on the basis of the measured recovery kinetics that assumed exponential repair. With the model, they were able to account for the $LD_{50/5}$ behavior with increasing exposure time and estimate a recovery half-time of 23.4 min.

Krebs and Leong also estimated that some 40 to 50 percent of the injury is irreversible. That accounts for an apparent maximum finite limit of slightly more than twice the minimum value approached by the $LD_{50/5}$ as the exposure rate becomes low in the absence of other repair mechanisms. However, this limit may be only transitory due to the onset of proliferation which might have been revealed for longer exposure periods. For example, as pointed out above, the lowest dose rate point (52 r/h) corresponds to an exposure period of 67 h (at least) or 2.8 days. In view that the fractionation studies of Withers and Elkind [1969] in mice provide clear evidence of proliferation

within that period, one would expect a correspondingly significant increase in the $LD_{50/5}$ associated with that dose rate (i.e., 52 r/h). Moreover, Withers [1972] gives evidence of crypt cell proliferation for 50 and 60 cGy/h based on constant dose rate exposures.

Travis et al. [1985] exposed mice to TBI from ^{60}Co radiation to assess the effect on lethality of constant dose rate, ranging from 60 to 1500 cGy. Death was scored at ten days to determine the dose rate dependence of deaths from the gastrointestinal syndrome. The $LD_{50/5}$ increased from about 1200 to 2050 cGy (about a factor of 1.7) for a decrease in dose rate from the high to the low ends of the range (1500 to 60 cGy/h). However, the $LD_{50/5}$ values are significantly lower by a factor of about 1.5 than those given by Krebs and Leong [1970] over about the same dose range. The difference could partially be attributed to different strains of mice used, although deaths scored at ten days by Travis et al. rather than within five days compared to Krebs and Leong, may have also been affected by damage to the hematopoietic system; pathologic findings by Travis et al. indicated changes in the jejunum, ileum, and rectum were minimal, and when they occurred subjectively, they appeared as a loss of crypts in both the jejunum and ileum. Also, in addition to source-to-target distance, lead shielding was used to reduce the source radiation to the desired exposure rates. This can produce a lower photon energy spectrum from scattering, resulting in an increase in RBE that would contribute to lower $LD_{50/5}$ values.

Dutreix et al. [1979] described studies of the effect on mice of constant dose rate exposures, ranging from 120 to 6000 cGy; they indicate a factor of 1.83 increase in $LD_{50/5}$, somewhat similar to that obtained by Krebs and Leong [1970] and Travis et al. [1985]. However, the actual $LD_{50/5}$ values are again significantly lower than those of Krebs and Leong over a corresponding dose range--a factor of about 1.6 lower at the high dose rate end and 1.8 lower at the low dose rate end. Again, some of the difference may be attributable to a different strain of mice, particularly in view that the same strain (Balb/c mice) used by both Dutreix et al. and Travis et al. were in agreement in LD_{50} values over corresponding dose rates and appeared to be well

within experimental error. Although not specifically indicated, presumably the LD_{50} values given by Dutriex et al. involved ^{60}Co irradiation. Whether or not shielding material was employed to attenuate source radiation exposures, which would change the photon spectrum, was not indicated.

Dutreix et al. [1979] also provided data to demonstrate the reduced effectiveness of protracted radiation in terms of a single acute dose equivalent of 10 Gy. The data are plotted in Fig. 33 for both constant dose rates (connected by the dashed line) and fractionated exposures (connected by the solid line). The time axis includes a three-hour interval which separates dose fractions all given in 10 min or less (the three-hour intervals allow adequate time for intracellular repair to take place) It is interesting to note that over the same period, the continuous dose rate exposure provides more protection than fractioned exposures since the acute single dose equivalent is a measure of residual damage. Also, the single acute

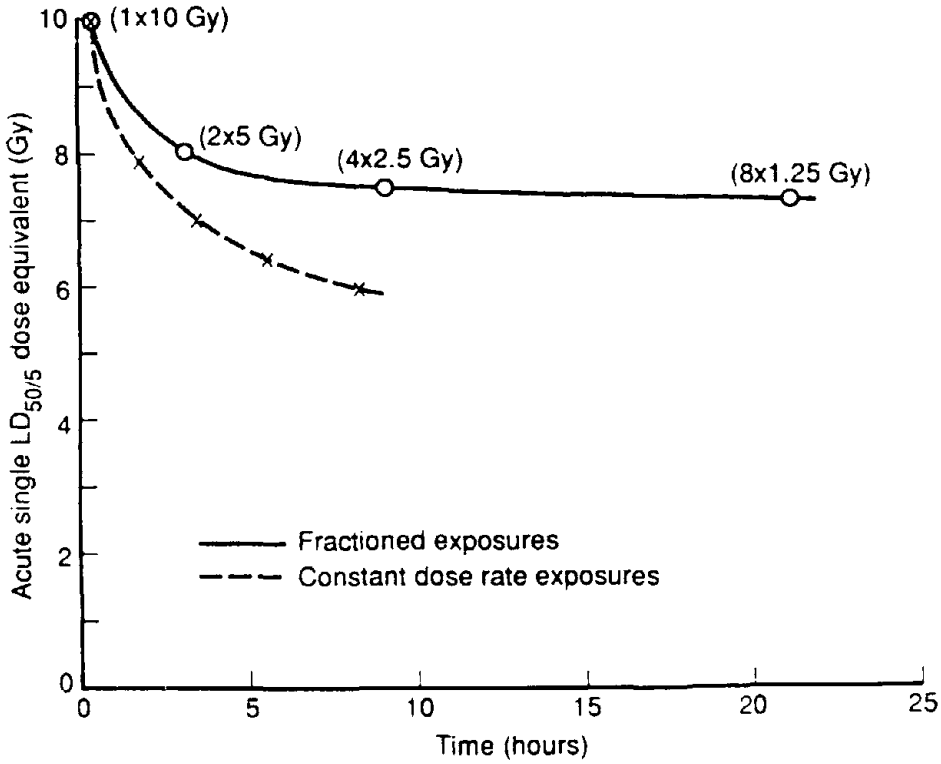
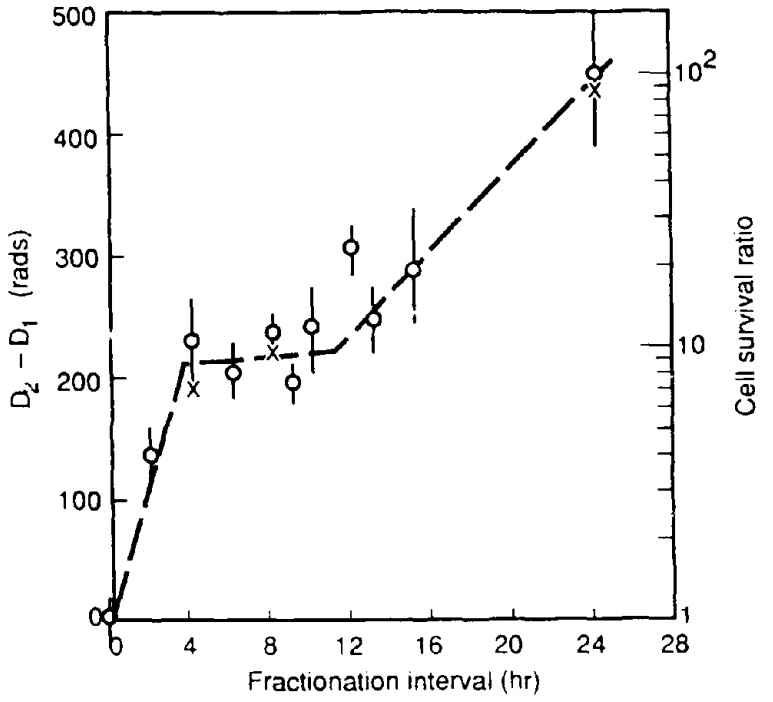


Figure 33. Acute single $LD_{50/5}$ dose equivalent to a 10 Gy protracted dose for constant dose rate and fractioned exposures.

dose equivalent appears to tend to an asymptotic limit with time (increasing number of functions) that would appear to be significantly higher than that for constant continuous radiation exposure. However, it would be difficult to assure this based on the limited data in Fig. 33. These data also point out that it would be inappropriate to infer the sparing effect of protracted radiation exposure based on dose-rate-averaging fractionated exposure. For example, 315 cGy/h would correspond to the dose rate obtained by averaging over the treatment time for the 2 × 5 Gy fractioned exposure where the acute single dose equivalent is larger than that for continuous exposure (i.e., 8 Gy compared to 7 Gy). Similarly, the dose rate averaged over the treatment time for the 4 × 2.5 Gy fractioned exposure is 109 cGy/h where the acute single dose equivalent is 7.5 Gy compared to only 6 Gy for continuous exposure.

Using 200 kVp X-rays, Withers and Elkind [1969] demonstrated intestinal radiosensitivity based on their milestone fractionated abdominal radiation studies in mice. They developed parallel data for the endpoints of lethality and jejunal crypt cell survival and determined that 50 percent animal survival within five days corresponds to a crypt cell survival fraction of around 0.002. Assuming a random crypt cell survival among the crypts, about 1/4 of the crypts will survive with proliferating cells. In normal mice, $LD_{50/5}$ (50 percent lethality at five days) generally corresponds to about 1/3 of the crypts surviving where stem cell survival is 0.003 [Withers, 1989]. Potten and Hendry [1983] indicate a higher value of 0.4 (range 0.3 to 0.5) for fraction of surviving crypts at the $LD_{50/5}$ for mice.

Fractioned radiation was given at a high dose rate (78 Gy/h) over two brief periods (5 to 11 minutes) separated by time intervals ranging from 2 h to 21 days. The first fraction was 660 rads followed by a second one of 1415 rads (total of 2075 rads) for *in vivo* cell colony counting. For lethality assessment, the second fraction was given to mice in graded amounts in order to calculate the $LD_{50/5}$ from probit analyses of data that straddled the $LD_{50/5}$. The single dose $LD_{50/5}$ was estimated to 1061 ±24 rads, and for split doses, it was always higher, as shown in Fig. 34.



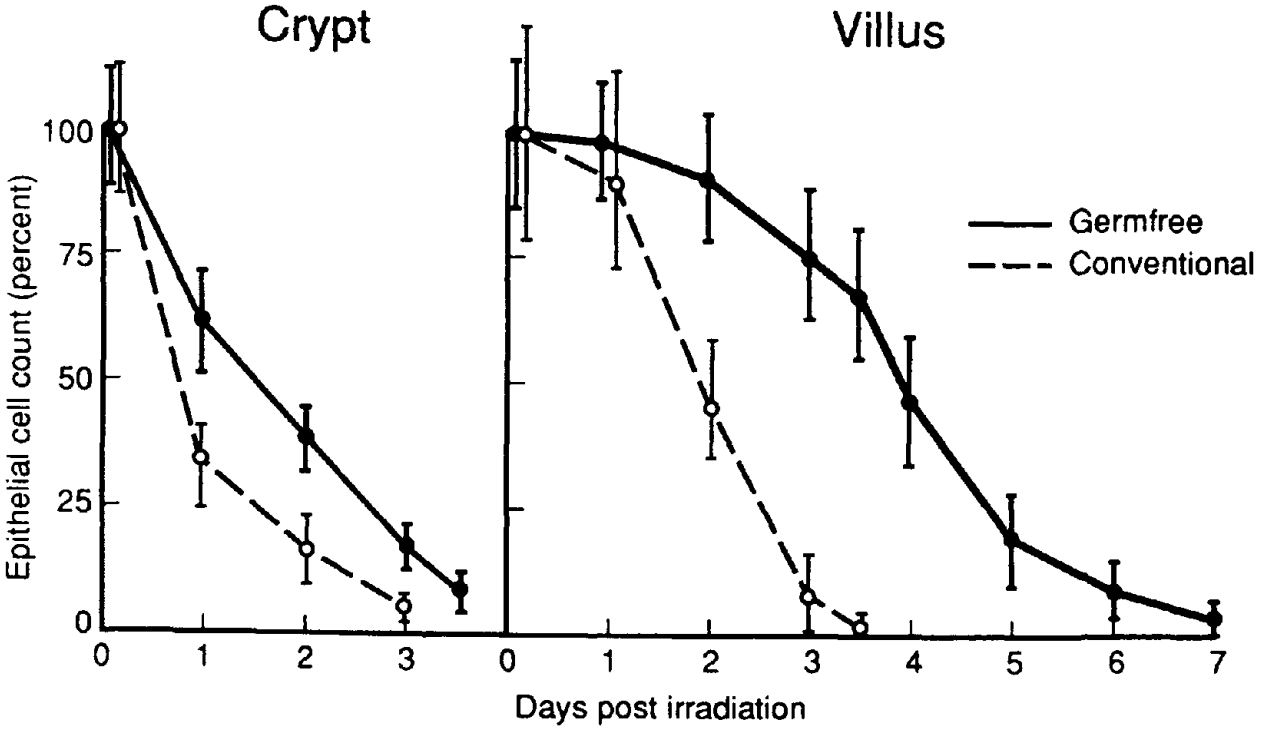
Source: Withers and Elkind [1969].

Figure 34. Recovery comparison of $LD_{50/5}$ (open circles and dashed line) and cell survival (x-symbols at 4-, 8-, and 24-hours) endpoints; the origin of both scales refer to 1060 rads acute single exposure.

The data points in Fig. 34 (open squares with standard error bars) along the dashed "eye-fit" line are the $LD_{50/5}$ data that correspond to the dose fractionation increment, D_2-D_1. In terms of lethality, it represents the increment of total dose required with increasing fractionation interval in order to maintain 50 percent lethality in mice at five days. The zero-dose increment value at the origin corresponds to a single acute dose of 1060 rads. The X-symbols lying dose to the $LD_{50/5}$ recovery curve at the 4-, 8-, and 24-h fractionation intervals are corresponding jejunal crypt cell survival ratios (right-hand ordinate) calculated at 1060 rads from expanded second-dose fraction cell survival data. The cell survival ratio of unity is at zero-dose increment that is normalized to the $LD_{50/5}$ value of 1060 rads for a single acute exposure (cell survival fraction is about 0.002). In terms of cell survival, the dose fractionation intervals represent the increments of total dose required with increasing fractionation interval in order to maintain a survival ratio

of unity (or the cell survival fraction at about 0.002). The similarity of the recovery data for cell survival and the $LD_{50/5}$ in Fig. 34 is evidence for a causal relationship between 5-day radiation death and lethal injury to stem cells of the intestinal mucosa.

Studies have been done using relatively high dose rates to characterize changes associated with intestinal radiation death or to determine possible causes. Matsuzawa and Wilson [1965] exposed mice to 3000 R of 250 kVp X-rays at a rate of about 40 R/min (~23 Gy/h) and determined a mean survival time of 3.5 days ranging from 2.9 to 3.8 days for conventional mice. For germfree mice, the mean survival was 7.3 days with a range of 6.4 to 7.7 days. Based on histological examination, they also obtained epithelial cell counts for the crypt and villi expressed as percentage versus post-irradiation time shown in Fig. 35. The curves show the progressive mucosal denudation with time that correlate well with lethality for both germfree and conventional mice. Based on the progress of thymidine -^{3}H labeled cells,



Source: Matsuzawa and Wilson [1965].

Figure 35. Cell counts of mouse ileum after a sterilizing dose of 3000 R.

cell transit times were estimated for conventional and germfree mice, given below.

	Transit Time (days)	
	Conventional Mice	Germfree Mice
Crypt to villi junction	0.5	1.6
Villi junction to tip	0.75	3.55
Total (crypt to villi tip)	2.1	4.3

Cell movement from the base to the tip of the villi was found to be linear with time. Also, both in the conventional and germfree mice, the normal transit time (i.e., approximate lifetime of the intestinal mucosal cells) is about 60 percent of the mean survival time. According to Matsuzawa and Wilson, the difference between the life span of the cells and time required for denudation (approximately mean time to lethality) indicates a change in the life span of the cells effected by a decrease in cell population.

Jackson and Geraci [1986] performed irradiation studies using conventional, pseudomonas-contaminated and GI-decontaminated rats to investigate the pathophysiological causes of radiation-induced gastrointestinal death. They employed fission spectrum neutrons from a TRIGA reactor and approximately 8 MeV average energy neutrons from an accelerator. Gamma irradiation was also done with ^{137}Cs radiation and ^{60}Co radiation at dose rates of about 25 to 26 Gy/h. They concluded that the inability of the denuded mucosa to absorb fluid and electrolytes and consequent hypovolemic shock was the major mechanism involved in producing intestinal radiation death.

Figure 36 and Table 20 show some results for $LD_{50/5}$ and median survival time, respectively, given by Jackson and Geraci. The RBE of cyclotron neutrons appears to be about two for gastrointestinal lethality regardless of treatment condition; for fission neutrons, the RBE is almost three (2.8). However, no significant difference was found in the median survival time for conventional rats regardless of the radiation source including gamma rays from either ^{137}Cs or ^{60}Co and neutrons from either the cyclotron or TRIGA. Also, the time of death is not explicitly dose dependent but given a sufficient amount of gastrointestinal damage (i.e., lethality threshold), the time of

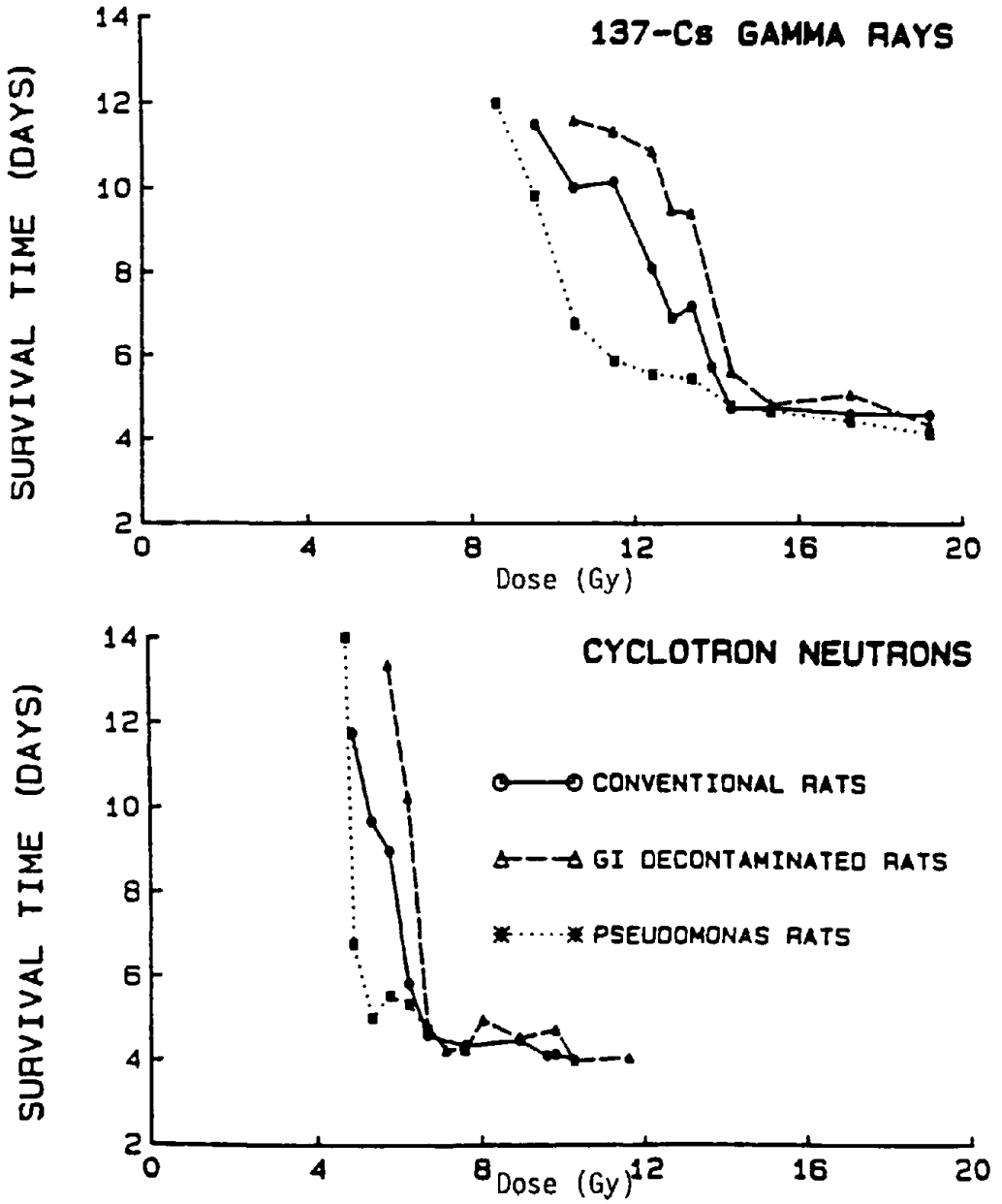


Figure 36. Survival time of untreated, GI-decontaminated, and Pseudomonas-infected animals after exposure to various doses of Cs-137 gamma rays and cyclotron neutrons (each point represents the median survival time of 8 to 40 rats). Source: Jackson and Geraci, 1986.

Table 20. $LD_{50/5}$ day of pseudomonas-infected GI, conventional, and GI-decontaminated rats.
Source: Jackson and Geraci, 1986

Treatment	Radiation	$LD_{50/5}$day (Gy)
Pseudomonas	137Cs γ rays	13.7 (13.1-14.3)[a]
Pseudomonas	C-neutrons[b]	5.85 (5.11-6.69)
Conventional	137Cs γ rays	14.1 (13.4-14.7)
Conventional	C-neutrons	6.25 (6.10-6.40)
Conventional	F-neutrons[c]	5.0 (4.72-5.30)
Decontaminated	137Cs γ rays	15.0 (13.9-16.2)
Decontaminated	C-neutrons	7.40 (6.79-8.09)

[a]Values in parentheses are 95 percent confidence limits.
[b]C-neutrons = Cyclotron produced neutrons.
[c]F-neutrons = TRIGA produced fission neutrons (1.2 MeV mean energy).

death proceeds according to degenerating physiological processes. When radiation exposure is protracted, the damage threshold is offset by recovery mechanisms which may either prevent or delay lethality.

Very little real data exist for lethality in humans that can be attributed purely to gastrointestinal injury. In fact, aside from being a predominate cause, it probably can never be expected to be the sole cause of lethality following TBI owing to other accompanying body injury. The most comprehensive set of lethality data for humans involving gastrointestinal injury is from the Chernobyl accident victims listed in Table 21 given by Guskova et al. [1988]. Dose estimates range from 10 to 12.5 Gy and the time of death from 10 to 20 days following the accident; the dose rates are indicated to be at a high level of at least 2 Gy/h or more [Baranov and Guskova, 1988]. Rather severe injury to the skin from beta radiation was a competing lethal effect as indicated in Table 21. The fact that some lethalities extended up to 20 days might reflect the extensive medical care given.

Table 22 gives estimates of dose and time of gastrointestinal-syndrome-lethality for man based on some pre-Chernobyl experience (with the exception of that indicated by Guskova et al., 1987). The

Table 21. Mortality of Chernobyl accident victims with skin and intestinal injuries.

Case Number	Dose (Gy) (Marrow)	Treatment[a]	Day of Death
3	12.0	BMT	17
4	11.8	BMT	18
10	11.1	FLT	14
14	10.9	FLT	18
15	>10.0	FLT	14
17[b]	10.0	BMT	18
20	12.4	FLT	17
23	13.7	FLT	15
26[b]	12.5		20
2097 (Kiev)	10.2		10

[a]BMT = bone marrow transplantation
FLT = fetal liver (cell) transplantation
[b]involved mycobacterial sepsis

accident cases given by Fanger and Lushbaugh [1967] are thought to be predominately due to gastrointestinal injury. Even though some of the dose ranges in Table 22 extend to 7000 cGy, Fanger and Lushbaugh [1967] give evidence for cardiovascular shock as the radiation

Table 22. Gastrointestinal syndrome lethality in man.

Dose (cGy)	Postexposure Time (days)	Source
1114, 1910	9, 10	Fanger and Lushbaugh (1967)[a]
1000-2000	8-16[b]	Guskova et al. (1987)
≥1000	<14	Maisin et al. (1971) [c]
>1000	7-10	Fajardo (1982)
670-6700[d]	7	Ingram (1969)
750-2000	7-14	Anno et al. (1989)
670-6700[d]	6-10	Lushbaugh (1973)

[a]Two accident cases cited from Hemplemann et al. (1952) and Kurshackov (1962).
[b]Intestinal changes most apparent, 8-12 days; total loss of epithelium, 10-16 days.
[c]Based on histologic examinations from autopsies performed on eight Hiroshima and Nagasaki bombing victims (substantial dose uncertainty)
[d]Based on a 0.67 internal-body-dose conversion factor for 1000-10,000 rads.

syndrome causing death based on pathologic changes in two accident victims who received doses of 4500 and 8800 cGy; furthermore, the times of death, 35 and 49 h, respectively, following exposure, are much too early for pure gastrointestinal injury to manifest in lethality.

We conclude that lethality solely from gastrointestinal injury in man exposed to TBI may exist in a limited range, perhaps ~8 to 12.5 Gy equivalent acute dose. Lethalities can be expected to occur within one to two weeks. However, GI injury may play an important part in lethality for doses on either side of that range. Also, when radiation exposure is protracted, the recovery potential of the intestinal mucosa can produce an increase in the total dose level required to effect lethality.

2.5 RADIATION INJURY AND RECOVERY.

Radiation injury and recovery research in mammalian species relevant to this effort is primarily based on the lethality endpoint from radiation-induced bone marrow cytopenia. The empirical techniques for the measurements required have been rather well established in a number of mammalian species. In other tissues, such as the intestines, *in vivo* injury and recovery assessment techniques with regard to the effect manifested in the whole organism have not been as well established. Accordingly, there exists a much larger body of literature with regard to demonstrating injury and recovery due to hematopoietic effects. However, compared to the hematopoietic system, the much earlier and more rapid rate of recovery of the intestines from radiation injury has been well established based on the work of many researchers including Quastler [1959], Quastler et al. [1959], Hornsey and Vatistas [1963], Lesher [1967], Withers and Elkind [1968, 1969, 1970], and Potten and Hendry [1975]. A more detailed discussion of intestinal injury and recovery accompanies the gut injury model we developed which is described in Section 5.

The postirradiation injury state, i.e., residual injury or injury that remains unrepaired, of an organism depends on the following considerations:

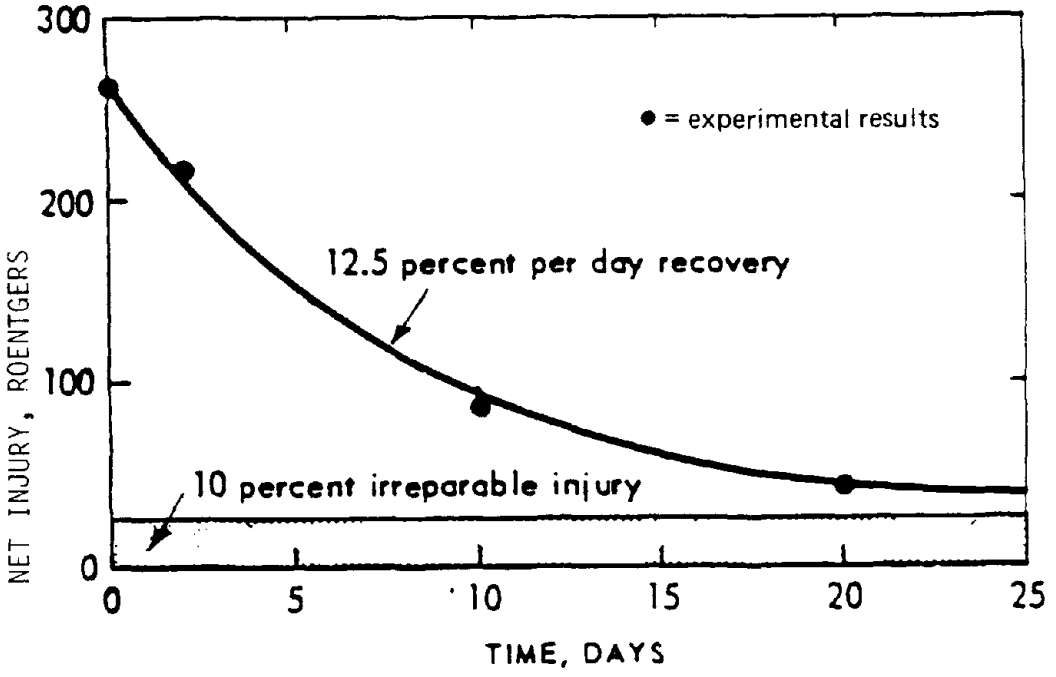
1. The extent to which the injury is dose dependent (i.e., radiosensitivity).
2. The physiological repair and recovery which can counteract the effects of injury.
3. Recovery must occur within a definite time or to a certain extent to effect survival.
4. Radiation exposure over a period of time induces fewer injuries per unit of time, permits repair processes to begin earlier, and enables proliferation to take place more rapidly.

Based upon lethality in mice from damage to the blood system, Blair [1952] developed a theory that radiation injury is repaired at a constant percentage per unit time of the net recoverable injury, and a small fraction of this injury is irreparable. This can be expressed in terms of net injury (acute single dose equivalent), D_e as

$$D_e = D_o [f + (1 - f)e^{-\beta t}]$$

where f = irreparable fraction of injury,
β = recovery rate in percent per day,
t = number of days,
D_o = single acute dose.

Davidson [1957] applied this equation to the results of a split-dose recovery study in mice by Patterson, Gilbert, and Mathews [1947]. He then derived the graph depicted in Fig. 37 where net or residual injury is expressed in roentgens. Davidson estimated 10 percent irreparable injury, and determined that 2.5 percent recovery per day would yield a curve that would best fit the data of Patterson, Gilbert, and Mathews. He then proceeded to estimate WBC recovery half-life for several mammalian species and correlated these data with minimum white cell counts in response to an LD_{50} exposure. He found an approximate linear correlation for the mouse, rat, dog,



Source: Davidson [1957].

Figure 37. Theoretical injury recovery curve with fraction of injury assumed unrecoverable.

and burro. Using human data available to him for WBC counts [Oughterson and Warren, 1956], Davidson estimated a recovery half-life for man of 690 h.

Subsequently, several investigators conducted a number of split-dose recovery and hematological studies to determine the validity of postirradiation exponential recovery and irreparable injury [Holloway et al., 1968; Ainsworth and Leong, 1966; Page et al., 1971; and Nachtway, Ainsworth, and Leong, 1967]. Irreparable or residual injury was confirmed; it appears to be dose, dose rate, and species dependent [Baum and Alpen, 1959; Baum, 1967]. Two examples of residual injury in the erythropoietic system are shown in Figs. 38 and 39. Rats were repeatedly irradiated with 300 or 400 R X-rays--five times at 90-day intervals. After each exposure, red cell production decreased during the recovery period. This decrease was significantly greater in rats subjected to 400 R X-radiation (Fig. 38). Figure 39 indicates that enthropoiesis significantly increases over the first five days following exposure.

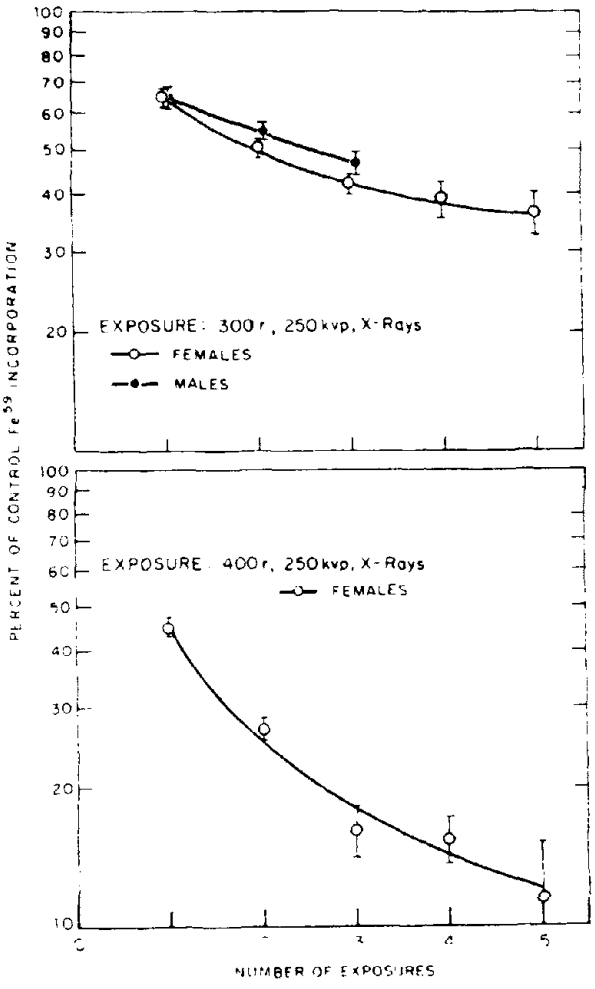


Figure 38. Maximum Fe^{59} incorporation for male and female rats exposed periodically to 300 r of X-rays and for female rats irradiated with 400 r (isotope was injected 5 days postirradiation, 3-month interval between exposures). Source: Baum and Alpen, 1959.

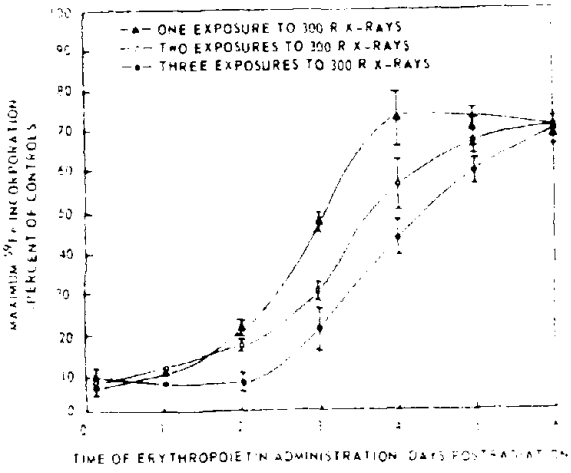


Figure 39. Residual injury of erythropoietic system in irradiated rats.

In Figs. 40, 41, and 42, data are plotted of split-dose recovery studies in hamsters and sheep performed at the Naval Radiological Defense Laboratory (NRDL) and rhesus monkeys at the Air Force School of Aerospace Medicine Laboratory [Holloway et al., 1968; Ainsworth and Leong, 1966; and Eltringham, 1967]. All these experiments show initial postirradiation recovery, over varying numbers of days, which is interrupted by a period of greater radiosensitivity (increased injury)

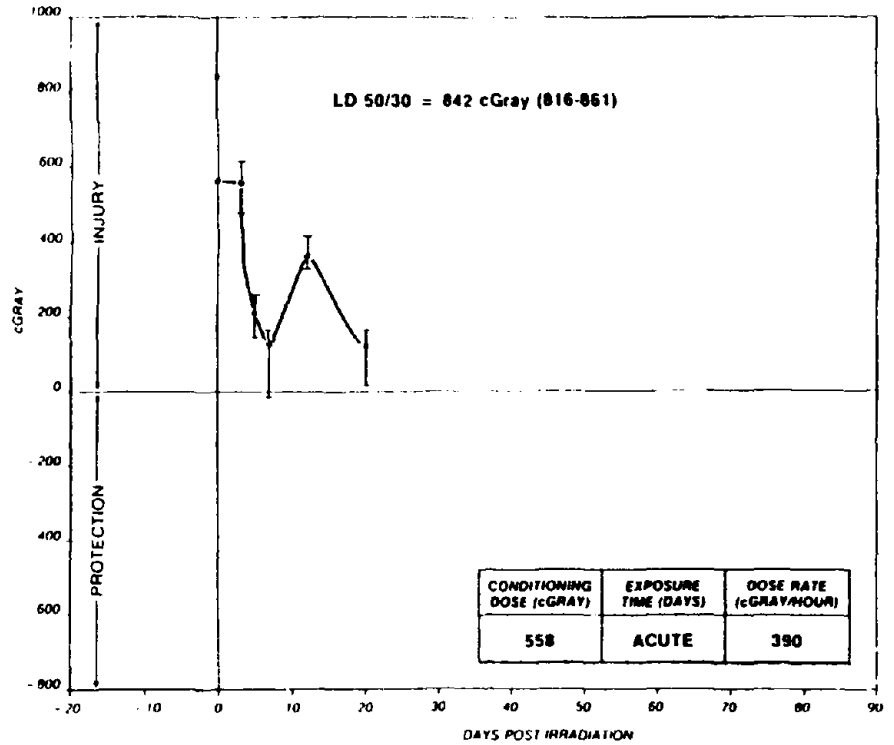


Figure 40. Hamster--acute exposure.

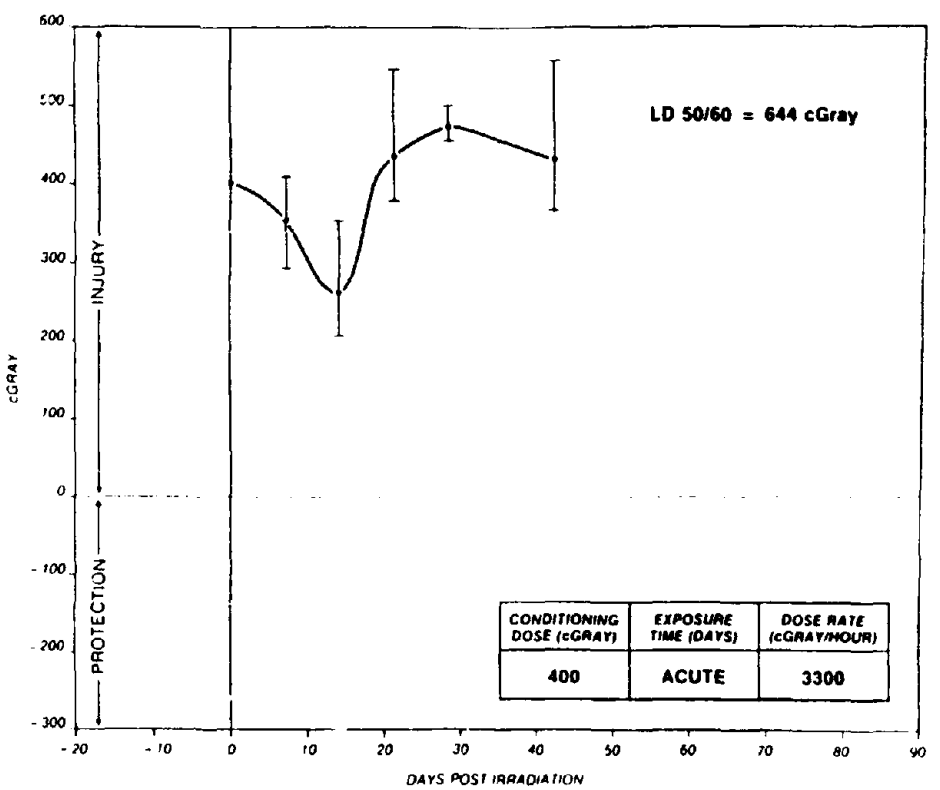


Figure 41. Rhesus monkey--acute exposure.

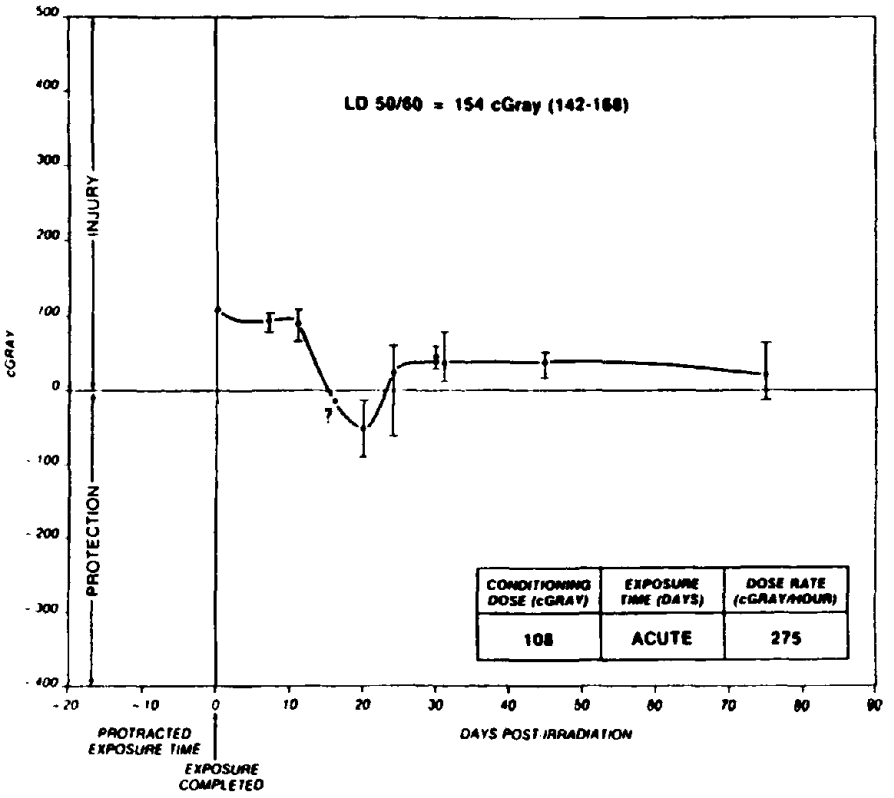
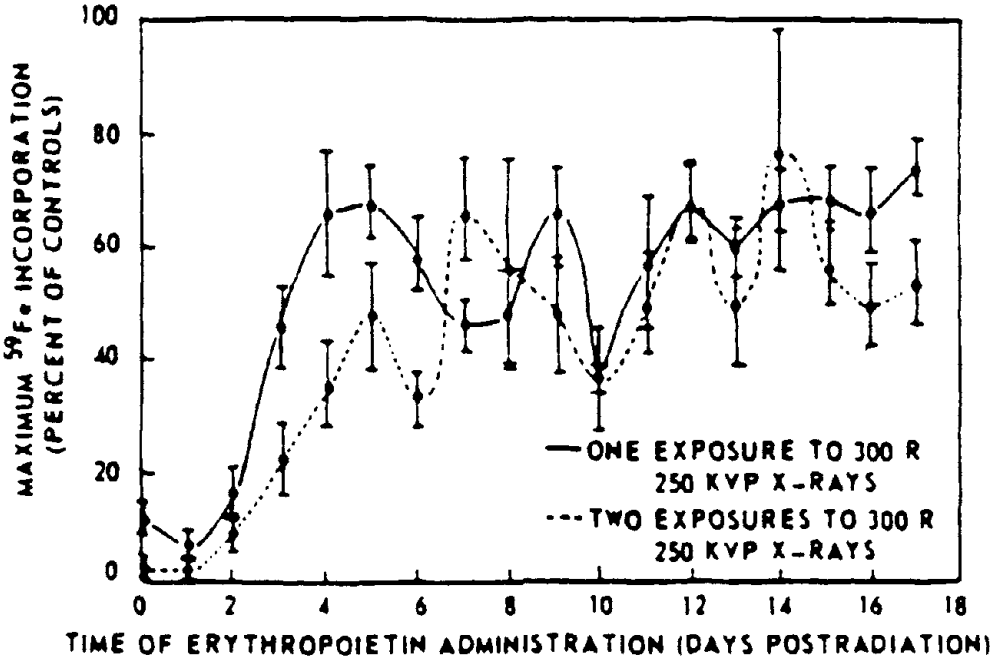


Figure 42. Sheep--acute exposure.

before final return to recovery occurs. None of these three species of animals appears to return completely to preirradiation levels. Similar reversals in recovery have been reported for the mouse and the rabbit [Leong, Wisecup, and Grisham, 1964].

Experiments designed to determine the pathophysiological responses of the blood-cell-forming system show postirradiation oscillatory recovery, which might be at least partly responsible for the return to hypersensitivity in the above-described split-dose recovery studies [Baum, 1967; Baum and Wyant, 1970; and Morley, King-Smith, and Stohlman, 1970]. Figures 43 (for the rat) and 44 (for the dog) demonstrate these oscillations for postirradiation red cell recovery. The finding that the above animals become radiosensitive after a period of substantial recovery suggests that a critical organ system (perhaps the bone marrow) has undergone some alterations such that little additional radiation is required to kill the animal. This, of course, questions the validity of Blair's theory of an exponential



Source: Baum (1967).

Figure 43. Residual injury of erythrocyte stem cells in irradiated rats--17-day observation period.

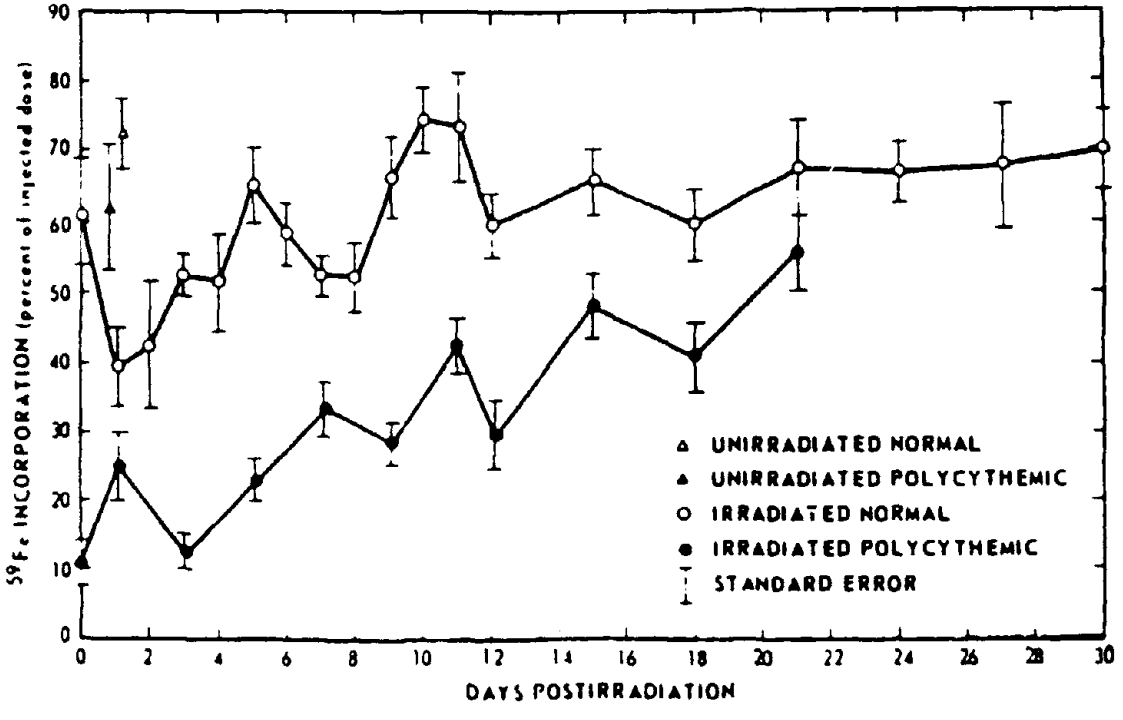


Figure 44. Erythropoiesis in normal and in polycythemic dogs exposed to 150 rads mixed gamma-neutron radiation. Source: Baum and Wyant, 1970.

recovery. Leong, Wisecup, and Grisham [1964] report a return to radiosensitivity for rabbits about three weeks after irradiation. Prior to that, continuous recovery of almost 60 percent was recorded. Since at the time of the reversed sensitivity, peripheral leukocytes were in the recovery phase and continued to recover, the authors questioned an involvement of the hematopoietic system with the return to radiation hypersensitivity. However, the postirradiation concentration of leukocytes in the circulatory blood system merely indicates that the perturbed bone marrow shunts newly formed leukocytes rapidly to the circulation for transportation to the tissues to protect against invading antigens. It does not necessarily mean that normal storage areas have been replenished and that sufficient numbers of white cells are available to counteract the deleterious effects of an additional dose of radiation.

In two other experiments (Figs. 45 and 46) conducted at NRDL using the dog and the swine, return to an increased injury condition is apparently not indicated. However, it must be emphasized that both studies were only carried out to day 20 postirradiation, and reversal of injury is seen in the sheep, another large mammal, between days 20 and 30.

For the swine, an experimental point is available for day 60 which could not be statistically evaluated. However, the swine may have a remarkable capability for recovery and be quite radioresistant. Figure 47 shows that swine exposed to an acute dose of 155 cGy given in about 0.5 h have sustained injury at the conclusion of the exposure equivalent to 155 cGy [Ainsworth et al., 1968]. If the same dose is given in 61.5 h, the remaining injury is only 64 cGy. Doubling or tripling the dose increases the injury only slightly. One possible interpretation of this finding is that swine develop increased resistance to radiation while being irradiated. Another may well be that animals sustain the greater part of their injury during the early radiation period. Experiments with sheep by Still et al., [1969] and Jones and Krebs [1970] seem to give credence to the latter interpretation.

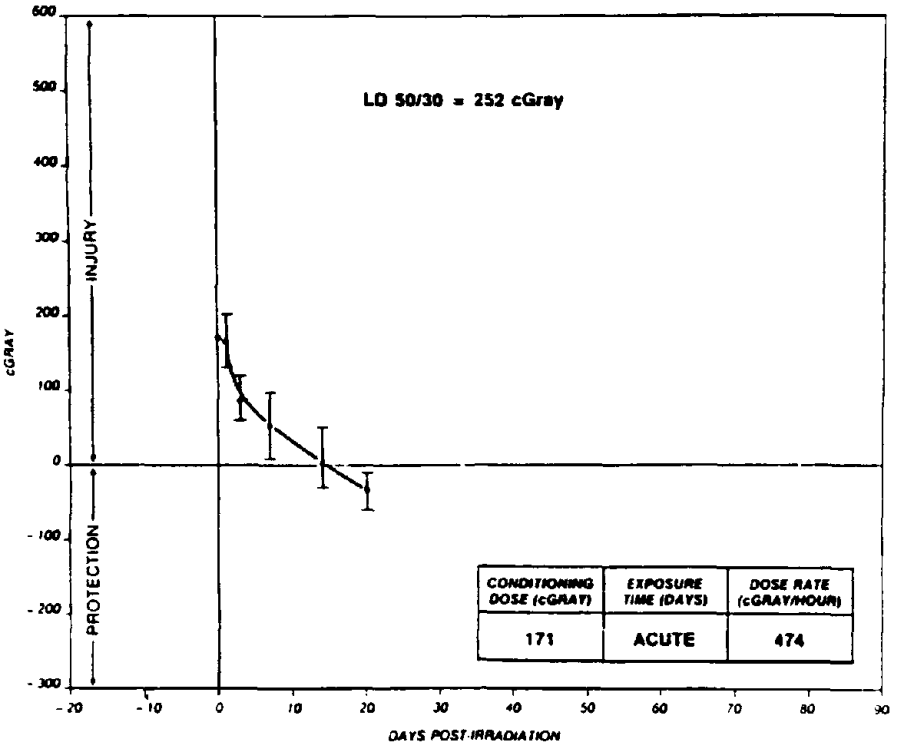


Figure 45. Dog--acute exposure.

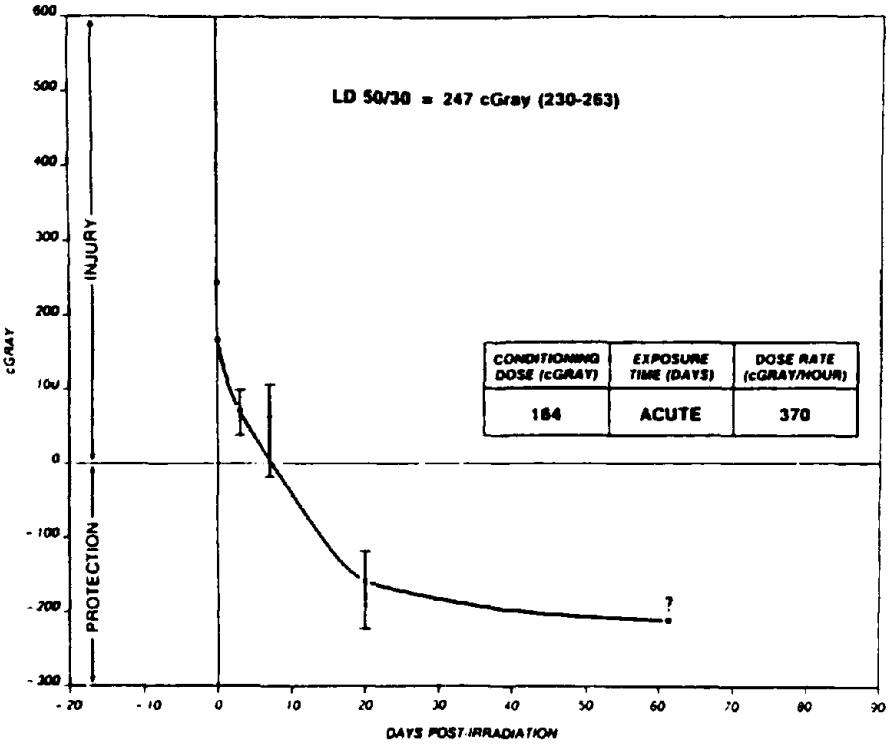


Figure 46. Swine--acute exposure.

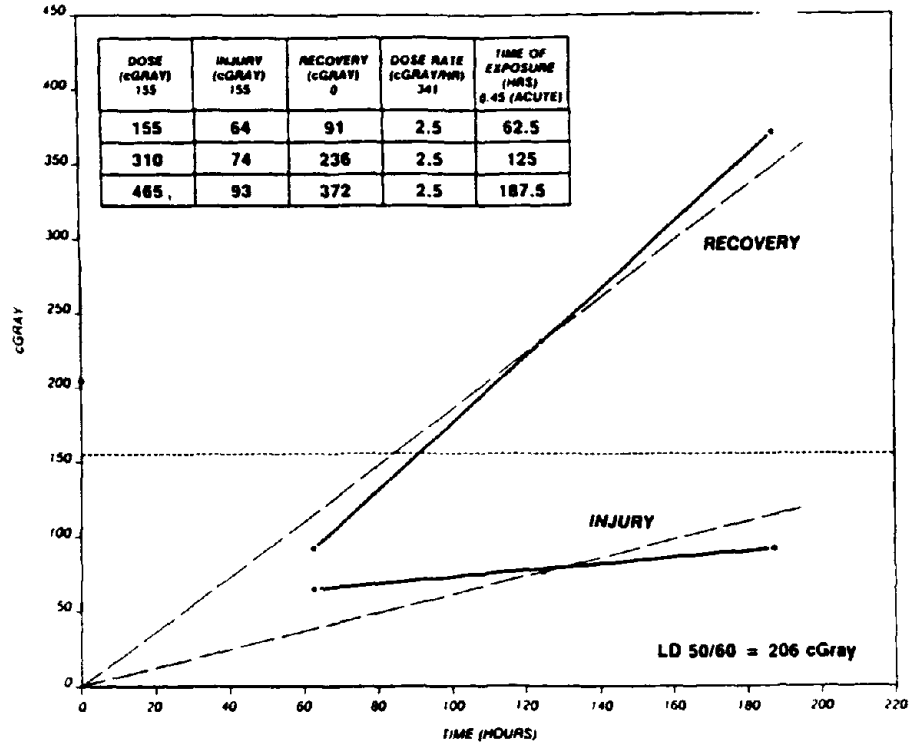


Figure 47. Swine--injury and recovery versus exposure time at fixed dose rate.

As indicated in Table 23, Still et al. [1969] gave animals 95 cGy at an acute dose rate of 311 cGy/h. Immediately after the completion of this conditioning (initial) dose, irradiation was continued at a reduced dose rate of 2.2 cGy/h until an LD_{50} of 199 cGy was obtained; this value was not significantly different from the $LD_{50/60}$ of 192 cGy observed when sheep received an acute dose rate at 275 cGy/h. However, 192 cGy did differ significantly from the $LD_{50/60}$ of 302 cGy obtained with a protracted dose rate of 2.2 cGy/h [Ainsworth et al., 1968]. The bottom half of Table 6 shows similar results observed in studies by Jones and Krebs [1970].

Figure 48 shows postirradiation recovery of five large mammalian species. The dog, sheep, and swine show similar recovery patterns over the first 20 days postirradiation, but otherwise, these curves are quite dissimilar. It is difficult to extrapolate a recovery pattern for humans from these curves unless other response parameters measurable in humans exposed to ionizing radiation can be correlated

Table 23. Protracted dose irradiation studies--sheep.

Experimental (Source)	Period												Total Dose (rads)
	(Initial Dose) 1			(Additional Dose) 2			(Final Dose) 3			4			
	Duration (h)	Dose Rate (rads/h)	Dose (rads)	Duration (h)	Dose Rate (rads/h)	Dose (rads)	Duration (h)	Dose Rate (rads/h)	Dose (rads)	Duration (h)	Dose Rate (rads/h)	Dose (rads)	$LD_{50/60}$
Still et al. [1969]	0.31	311	95	47	2.2	104							199
Still et al. [1969]	0.7	275	192										192
Still et al. [1969]	0.35	403	145										145
Still et al. [1969]	42	2.4	101	0.2	403	81							182
Ainsworth et al. [1968]	137	2.2	302										302
Jones and Krebs [1970]	0.45	353	158										158
Jones and Krebs [1970]	0.08	348	27	35.6	2.3	82	0.16	348	56				165
Jones and Krebs [1970]	0.46	350	160										160
Jones and Krebs [1970]	0.02	348	5.6	35.6	2.3	82	0.24	348	82.4				170
Jones and Krebs [1970]			0	35.6	2.3	82	0.29	348	100				182

to them; reevaluation of postirradiation leukocyte responses in man might be helpful.

Sheep are the only large animals for which data are available to compare the radiation effects of various low dose rates with an acute one. Such studies were conducted at NRDL, and the results are depicted in Figs. 49 to 53. As may be seen from Table 24, the first five conditioning doses employed were similar. It appears that when sheep are exposed at below a dose rate of 1 cGy/h, no apparent injury remains at the end of irradiation. Above a dose rate of 1 cGy/h, injury can be determined at the end of the radiation exposure, which appears to increase with increasing dose rate. As the conditioning dose increases (186 cGy in Table 24), the remaining injury increases in direct proportion. When the conditioning dose is 101 cGy, injury accumulates at 0.63 cGy/cGy. However, when the total dose is increased to 186 cGy at the same dose rate, injury accumulates at only 0.5 cGy/cGy. Maximum over-recovery, given a similar size of conditioning dose, increases with dose rate and appears to be proportional to remaining injury, at least over the dose rate ranges indicated in Table 24. It appears that the severity of initial injury may be related to a humoral or hormonal release that stimulates increased cellular production in recovering bone marrow.

It has been established that recovery and repair are important factors that enable mammals to sustain increased radiation injuries with protracted exposures. Split dose experiments permit the measurement of recovery based on the increase in the radiation dose to satisfy a specific endpoint (e.g., 50 percent lethality). More complete measurements obtained from split-dose experiments could facilitate the development of relationships for dose protraction in animals and eventually extrapolations to humans. If physiological processes cause a deviation from a simple recovery process such as a return to hyper-radiosensitivity after a period of normal recovery (i.e., an undulating pattern), the underlying pathophysiology of the system which induces such changes must be considered. Cell cycling sensitivity to irradiation exposure, as demonstrated by Bedford et al. [1980], may also play a part, which should also be among the underly-

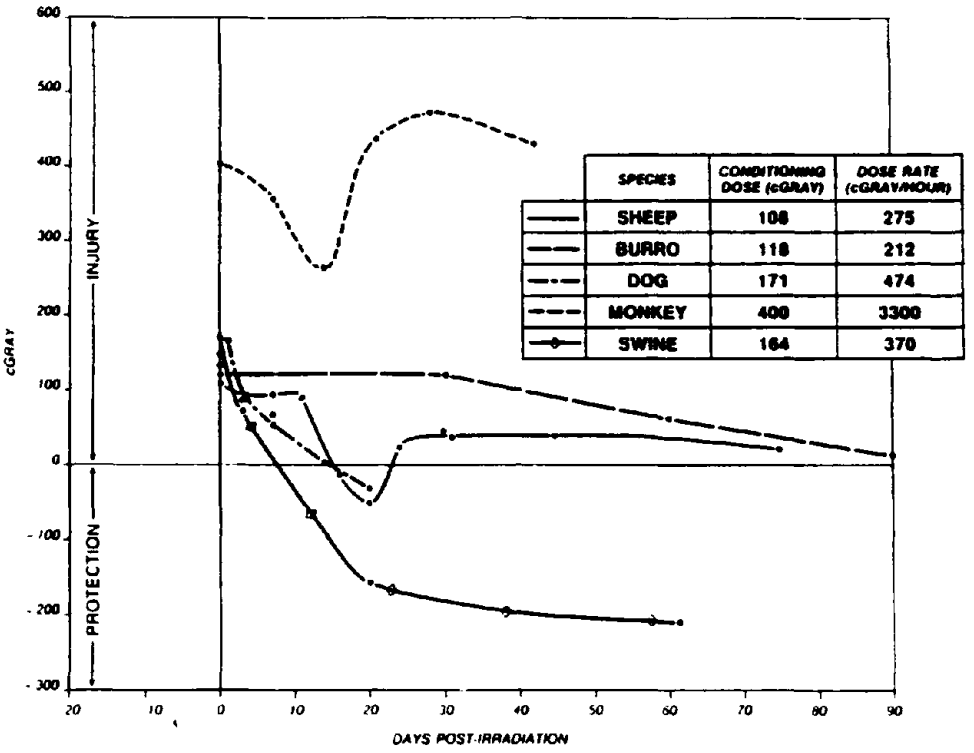


Figure 48. Species comparison for acute exposure.

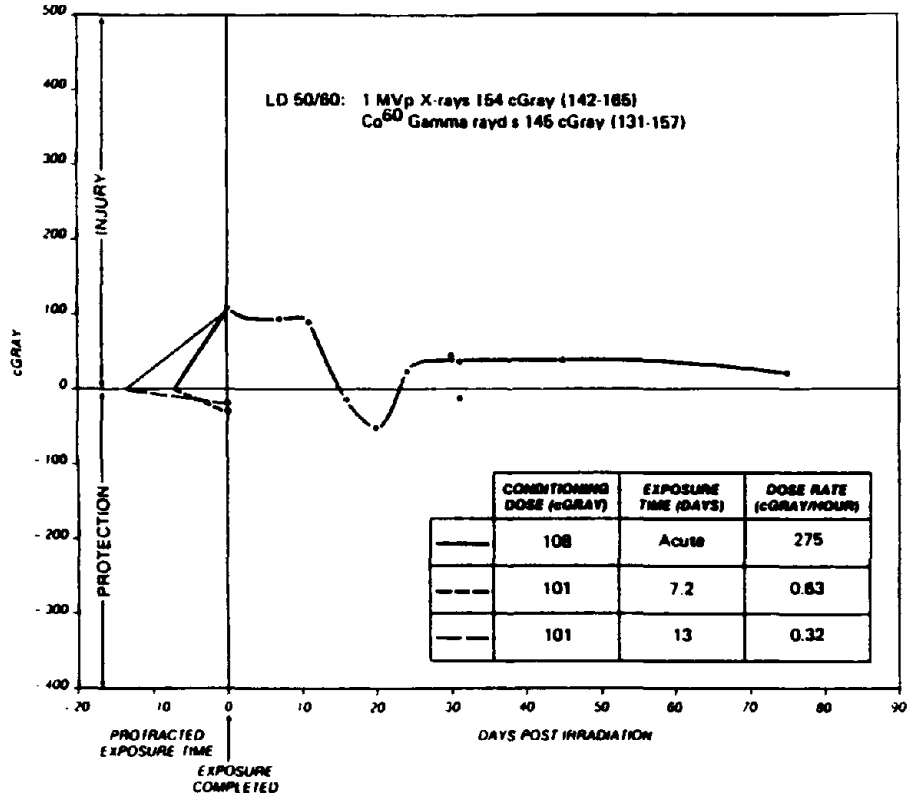


Figure 49. Sheep--acute and protracted dose (0.63 and 0.32 cGy/h).

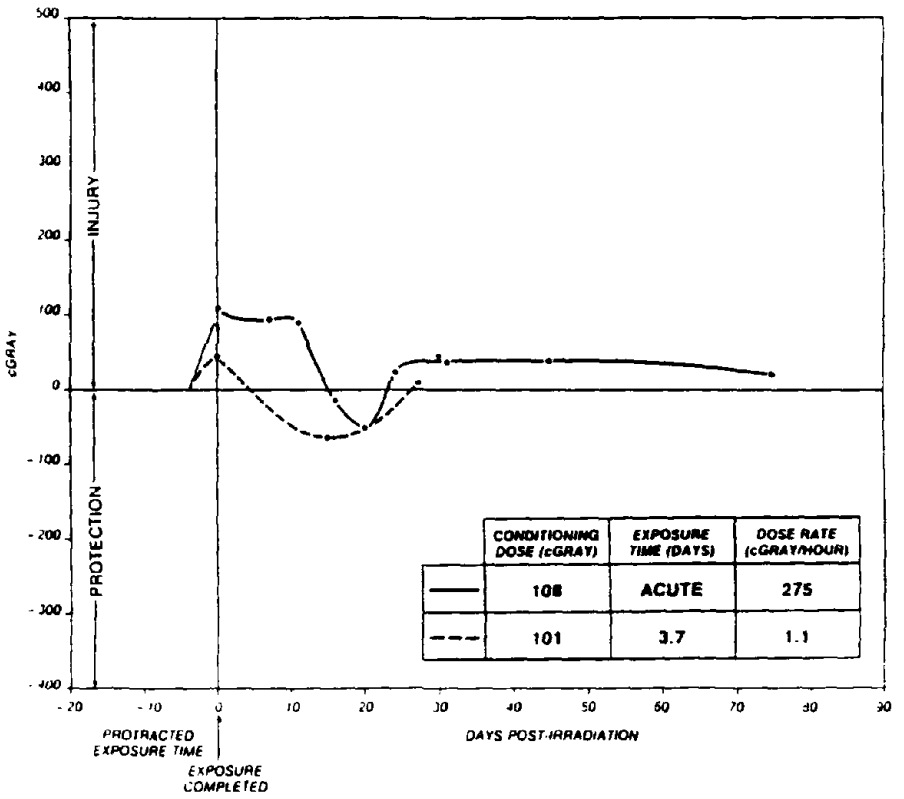


Figure 50. Sheep--acute and protracted dose over 3.7 days (1.1 cGy/h).

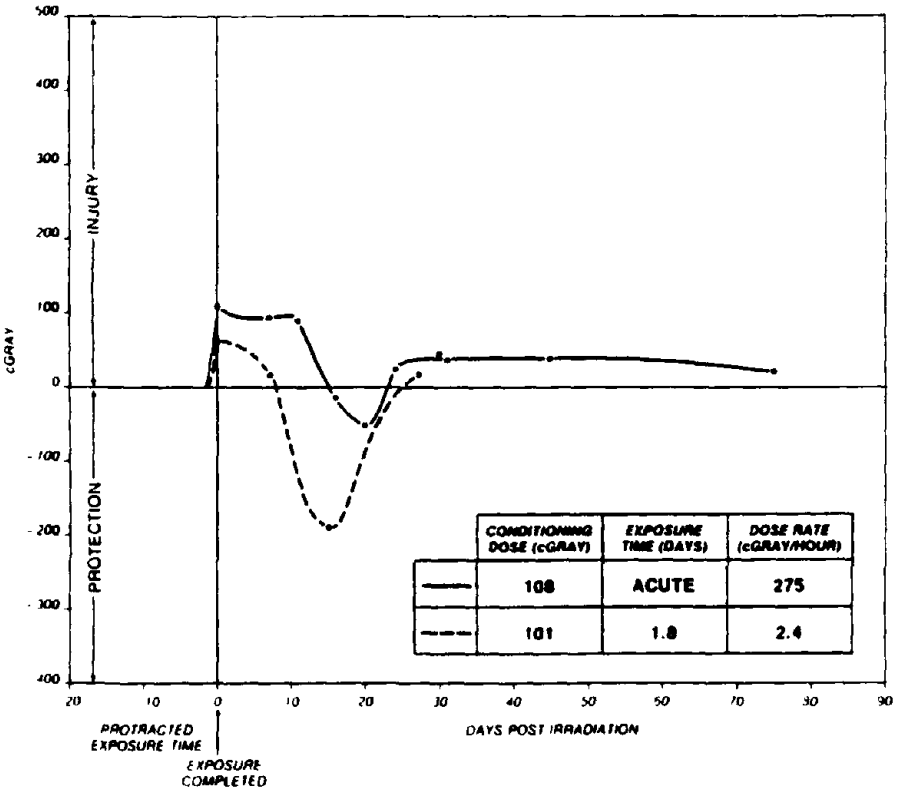


Figure 51. Sheep--acute and protracted dose over 1.8 days (2.4 cGy/h).

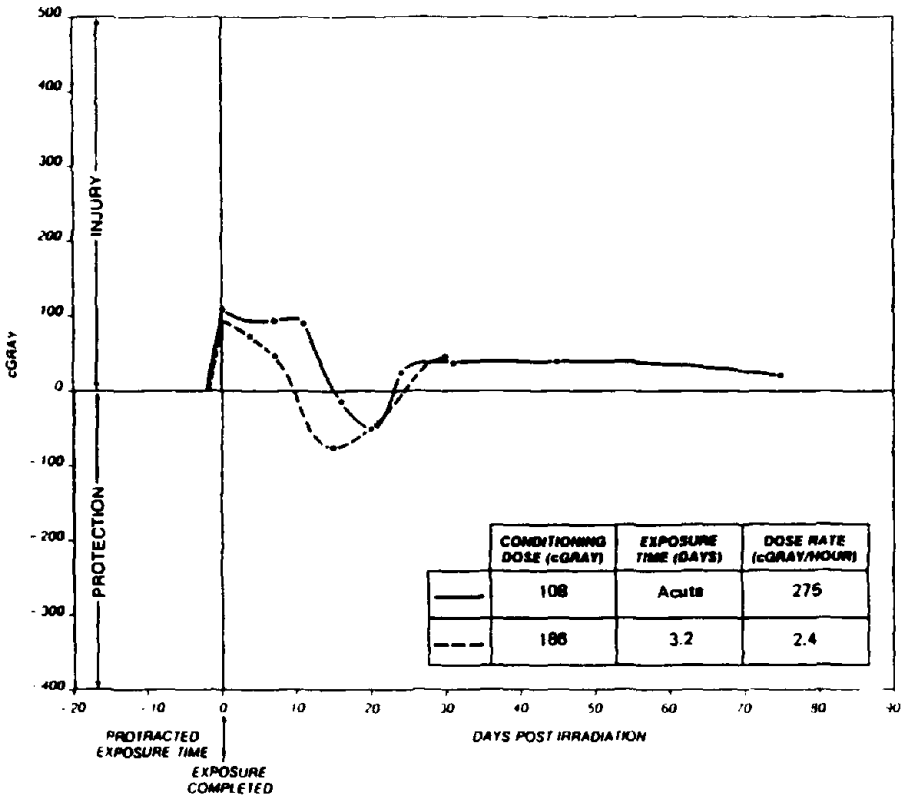


Figure 52. Sheep--acute and protracted dose over 3.2 days (2.4 cGy/h).

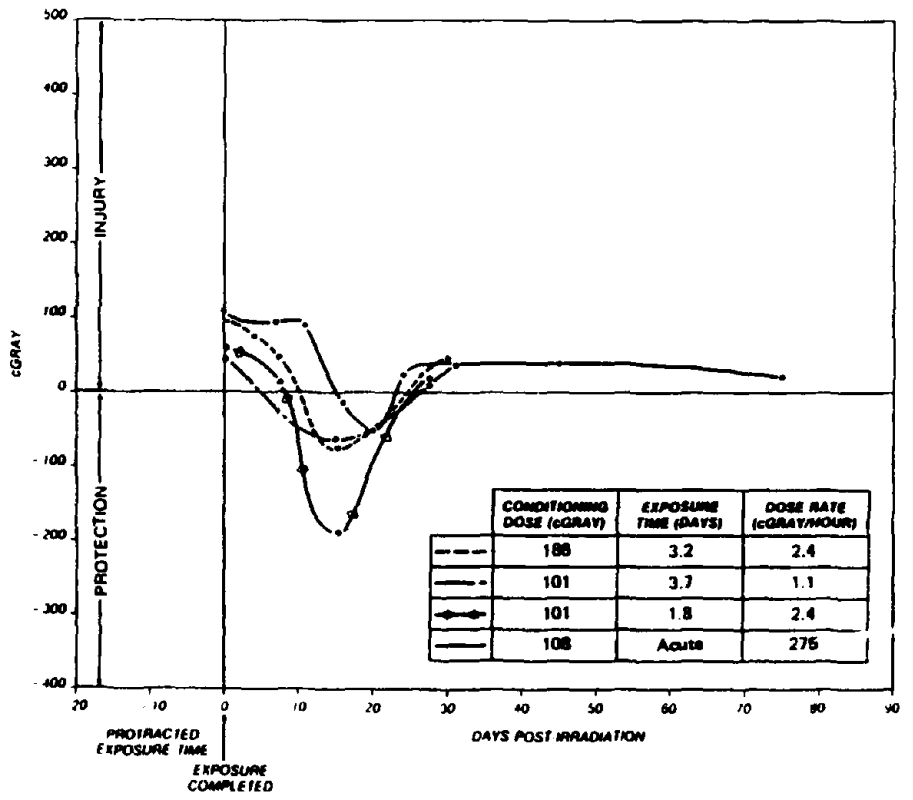


Figure 53. Sheep--acute and protracted dose comparisons.

Table 24. Remaining injury and maximum over-recovery in sheep subjected to radiation at different rates.

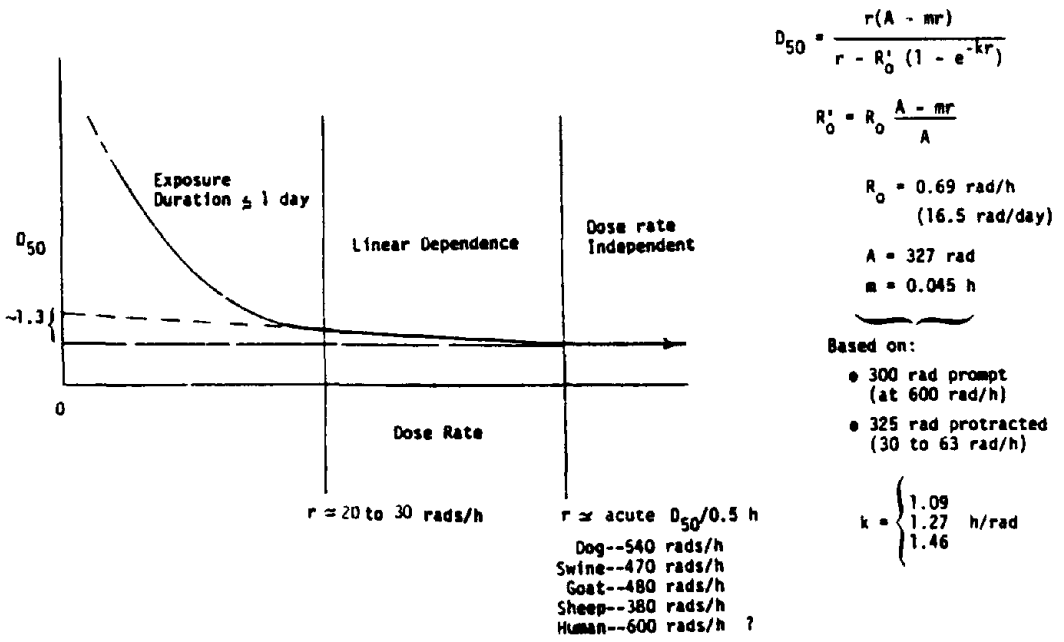
Conditioning Radiation Dose (cGy)	Dose Rate (cGy/h)	Exposure (h)	Time (days)	Remaining Injury (cGy)	Maximum Over-recovery (cGy)
108	275	0.4	--	108	-44
101	0.32	316	13	0	-19
101	0.60	168	7.2	0	-26
101	1.1	92	3.8	46	-64
101	2.4	42	1.8	64	-188
186	2.4	77.5	3.2	93	-75

ing casual factors considered.

2.5.1 Constant Dose Rate Models.

Accumulated injury models were reviewed by Anno and Baum [1986] in terms of LD_{50} endpoint as a function of constant dose rate level such as depicted in Fig. 54. Figure 54 also summarizes some of the findings of Krebs and Jones [1975] from their review of animal data (sheep, dogs, swine, goat, and mice) and suggestions for modeling LD_{50} response in animals, including extrapolation to humans. They developed a relationship LD_{50} (D_{50} in Fig. 54) as a function of dose rate r, which includes linear and exponential relationships, to model protracted radiation response. In their analysis of the animal data, Krebs and Jones found, in part, that

- lethal dose becomes dependent upon dose rate when the time required to deliver it is longer than about 30 min (otherwise, the LD_{50}-dose-rate relationship is flat where dose rates can be considered "acute"),



Source: Krebs and Jones [1975].

Figure 54. LD_{50} versus dose rate.

- the LD_{50}-dose-rate relationship is linear between about 20 to 30 rads/h and the acute dose rate level,
- between about 2.5 rads/h and 20 to 30 rads/h, there is a transition from a linear LD_{50}-dose-rate relationship to an exponential one,
- less than about 2.5 rads/h, the LD_{50} depends only on the average daily dose rate which can be averaged out over as much as two weeks.

Anno and Baum [1986] compared accumulated human injury models by plotting LD_{50} (rads) versus dose rate r (in rads per h). That comparison, shown in Fig. 55, required some algebraic adjustments to obtain the appropriate plotting forms that are given below along with the parameters.

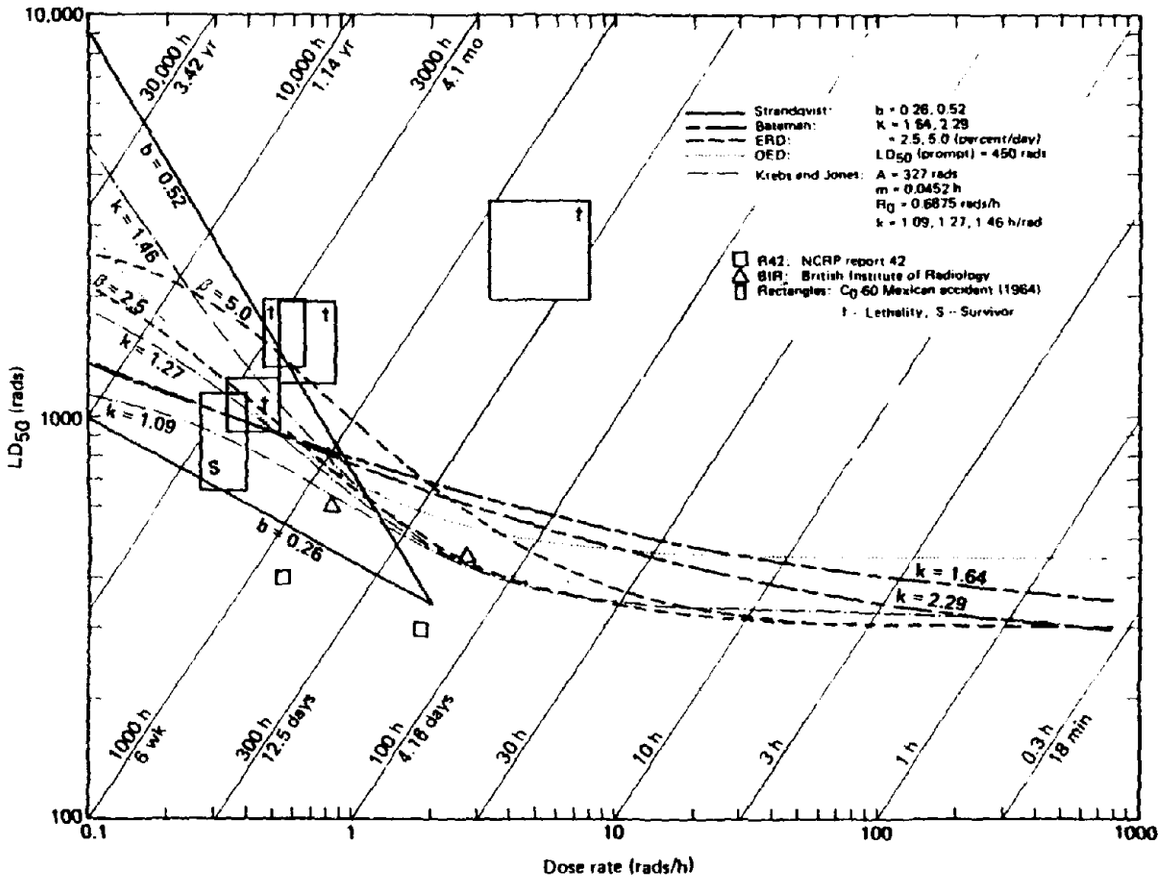


Figure 55. LD_{50} dose rate models.

Strandqvist [1944].

$$D_{50} = D_o^{[1/(1-b)]} \cdot (168r)^{-[b/(1-b)]}$$

where D_o = 345 rads (acute LD_{50}) and b = 0.26, 0.52.

Bateman [1968].

$$D_{50} = D_o(1 + Kr^{-1/3}) \quad ,$$

where D_o = 300 rads (fixed), K = 1.64 $(rads/h)^{1/3}$ based on the 1964 Mexican accident [Martinez et al., 1964]; and D_o = 236 rads, K = 2.29,

also based on the 1964 Mexican accident, but with the acute dose anchored at D_o = 300 rads for r = 600 rads/h.

Equivalent Residual Dose [Blair, 1952; Davidson, 1957].

$$D_{50} = \frac{1}{f}\left\{ERD - \frac{(1 - f)}{\beta}\left[1 - \exp(-\beta D_{50}/r)\right]\right\} \quad ,$$

where ERD (equivalent residual dose) = 300 rads, f (irreparable fraction) = 0.1, and the repair constant β = 0.00104 h^{-1} and 0.002083h^{-1}; (for β = 2.5 and 5.0 percent per day, respectively).

Operational Equivalent Dose, [Home Office Scientific Research and Development Branch, 1985].

$$D_{50} = OED + \frac{250}{r} \quad ,$$

where OED (operational equivalent dose) = D_{50} (acute) = 450 rads.

Data from other sources are also individually plotted in Fig. 55. The two box-shaped "R42" values are based on the LD_{50} values given in the "Penalty Table" by the National Committee on Radiation Protection and Measurements [1974]. For one-week exposure, an LD_{50} of 300 rads (450 R) is given; that corresponds to an average dose rate of about 1.77 rads/h (2.68 R/h). For one-month exposure, an LD_{50} value of 400 rads (600 R) is given; that corresponds to an average dose rate of about 0.55 rads/h (0.82 R/h).

The two values marked "BIR" in Fig. 55 are based on information from the British Institute of Radiology (BIR) as quoted on p. 84 in the British Medical Association report of 1983.

The rectangles in Fig. 55 reflect dose and dose rate uncertainty and are based on the 1964 Mexican accident involving cobalt-60 γ-ray radiation exposure of five family members [Martinez et al., 1964] that resulted in four deaths (†) and one survivor (S). Accordingly, these are not LD_{50} data, but are shown for reference only.

Plots of the protracted radiation response models (Fig. 55) show a considerable variation in accumulated lethal exposure dose versus dose rate. However, with the exception of the Bateman model, those plotted suggest a marked increase in LD_{50}, commencing with dose rates less than about 3 to 10 rads/h; the even more rapid increase in LD_{50} for dose rates from about 1 to 3 rads/h probably reflects cell proliferation. Anno and Baum [1986] provide a detailed discussion of the model plots.

A brief review of some suggested lethality-endpoint-based models of protracted radiation response illustrates the need for additional investigation. Lethality is only one of the endpoint responses of interest in casualty considerations; however, the models do predict various degrees of biological recovery. Any model selected should be better substantiated by more in-depth analysis of available data from animal studies and preferably human experience; for example, the Goiania, Brazil accident involving protracted exposure to cesium-137 γ-ray radiation from an abandoned teletherapy unit [International Atomic Energy Agency, 1988] could yield more clinical information. At present, data on arbitrary exposure periods and/or varying dose rates are scarce or limited in scope. Consequently, our comparisons of the protracted radiation exposure models are based on continuous and constant exposure rate levels. However, as Krebs and Jones [1975] imply, when average daily dose rates are less than about 2.6 rads/h (or about 62 rads/day), the exposure history for the 24-h period is largely irrelevant.

Our investigation of the kinds of models reviewed here indicates that they cannot be generally applied to other endpoints such as prodromal responses without possibly data-supported modifications--and then only for constant dose rate exposure. However, such models have been applied for prodromal symptomatology of protracted radiation involving fallout effects analysis [Knapp, 1965; and Schmidt, 1981]. Because the kind of biological recovery illustrated in this review may not adequately model other processes (such as a physiological clearing action and recovery), a different type of modeling approach is generally necessary to accommodate prodromal responses to protracted

radiation. For example, for dose rates in the therapy range of about 1 to 30 rads/min (60 to 1800 rads/h), there are indications that nausea and vomiting depend more on the total accumulated dose than on the dose rate [Baum et al., 1984]. Our assessment of the existing models of protracted radiation based on lethality as the endpoint reveals that additional study of available data is needed before proceeding to a systems analysis approach that is used in military operations and planning.

2.5.2 Operational Equivalent Dose as Injury Accumulation.

The residual injury accumulation based on the OED formula recommended for application in the UK [Home Office Scientific Research and Development Branch, 1985] is illustrated in Fig. 56. The OED formula is given by

$$OED = D - 150 - 10t \quad \text{(rads)} ,$$

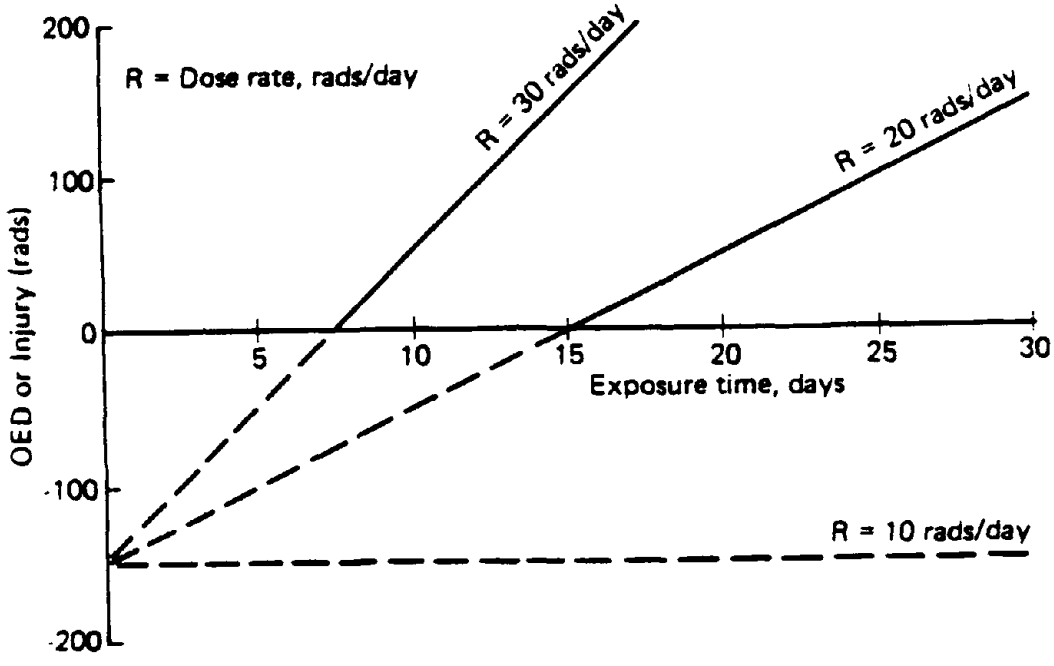


Figure 56. OED (injury, for constant daily exposure rate.

where D refers to the accumulated dose to the bone marrow, t is the exposure time in days, 150 (rads) is a rapid (Elkind) repair or recovery value that takes place within the first day following exposure, and 10 rads/day represents a daily recovery rate. Given a dose rate R, and letting D = Rt, the OED (or accumulated injury) can be expressed as

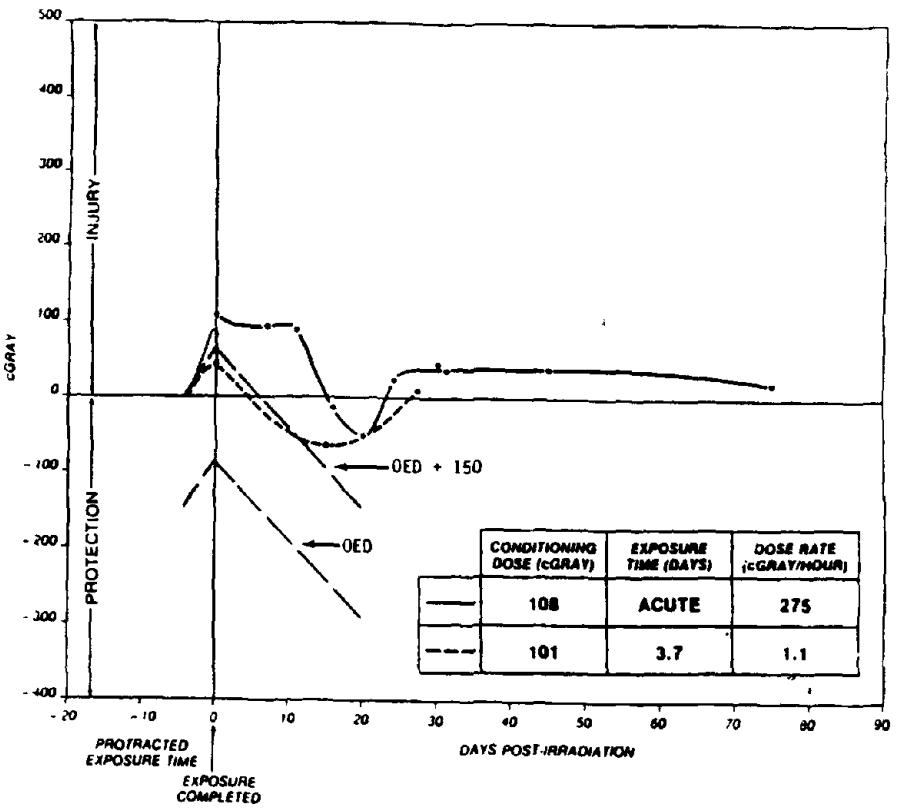
$$OED = (R - 10)t - 150 \quad \text{(rads)} .$$

Plots of the OED relationship above for dose rates, R = 10, 20, and 30 rads/day are shown in Fig. 56. Since negative values are ignored in applying the OED formula, the residual injury would remain at zero until it accumulates linearly after specific exposure times, depending on the dose rate, as shown.

The OED was also compared to data derived from experiments with sheep [Hanks et al., 1966]. Figure 57 illustrates the comparison for radiation exposure protracted over 3.7 days at the rate of 1.1 rads/h or 26.4 rads/day. The initial portion of the bottom curve (large dashes, marked OED) is a plot of the OED relationship given above over the period of exposure. For postirradiation times where R = 0, it is assumed that recovery continues at the rate of 10 rads/day.

It is clear from Fig. 57 that the OED considerably overestimates recovery from injury compared to the data drawn from experiments with sheep. The overestimate is primarily due to the 150 rads that purportedly accounts for "rapid recovery." When the 150 rads of repair recovery is ignored, given by the curve marked OED + 150, the agreement with the experimental data is vastly improved out to about 12 days postirradiation. This underscores the most significant problem with the OED formula, although the recovery rate of 10 rads/day appears to be within reason.

Our review and analysis of the literature provided us with a comprehensive assessment of radiobiological injury and recovery relevant to acute and protracted radiation exposure. In the section that follows, we discuss aspects of the review key to the development of modeling approaches for upper and lower gastrointestinal distress.



Source: Hanks, et al. [1966] for data on sheep.

Figure 57. Residual injury--comparison between formula and data derived from experiments with sheep.

SECTION 7

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APPENDIX A

UGIDM EQUATIONS FOR ACUTE AND CONSTANT DOSE RATE EXPOSURES

This appendix derives solutions of the UGIDM equations for two specific cases of radiation exposure: acute (or prompt) and constant dose rate. The model variables and parameters are:

Variables

- C = depletable reservoir (target tissue) level, Gy
- P = potential toxin, Gy
- A = active toxin, Gy
- R = dose rate, Gy/h^{-1}
- D = dose, Gy

Parameters

- α = potential toxin conversion rate/active toxin production rate, h^{-1}
- β = active toxin clearing rate, h^{-1}
- μ = depletable reservoir reconstitution rate, h^{-1}
- C_o = initial reservoir level, Gy
- D_o = characteristic target tissue dose ($=C_o$), Gy
- $A_{0.5}$ = half-maximum value of A, Gy
- γ = severity response slope

A.1 ACUTE EXPOSURE.

The UGIDM differential equations for acute exposure are,

$$\text{depletable reservoir:} \quad \frac{dC}{dt} = \mu\left(C_o - C\right) \tag{A.1}$$

$$\text{potential toxin:} \quad \frac{dP}{dt} = -\alpha P \tag{A.2}$$

$$\text{active toxin:} \quad \frac{dA}{dt} = \alpha P - \beta A \quad . \tag{A.3}$$

Following an acute exposure, the initial amount of potential toxin derives from the depletable reservoir in the UGIDM. This can be determined by taking the appropriate limits of the solution to the differential equation for the depletable reservoir at a constant dose rate, R, given by,

$$\frac{dC}{dt} = - \left(\frac{R}{D_o}\right) C + \mu\left(C_o - C\right) \quad , \tag{A.4}$$

or $$\frac{dC}{dt} + KC = \mu C_o \quad , \; K = R/D_o + \mu$$

and then, $$\frac{d}{dt}\left(Ce^{Kt}\right) = \mu C_o e^{Kt} \quad .$$

Integrating, $$\int_{C_o}^{Ce^{Kt}} d\left(Ce^{Kt}\right) = \mu C_o \int_o^t e^{Kt} dt \quad ,$$

and solving for C(t) gives,

$$C(t) = \frac{\mu C_o}{K}\left(1 - e^{-Kt}\right) + C_o e^{-Kt} \tag{A.5}$$

Taking limits of the dose rate (R) and time (t) along the fixed relationship, $D = Rt$ (where $R/D_o \gg \mu$),

$$\lim_{\substack{R \to \infty \\ t \to 0}} C(t) = C_o e^{-D/D_o} \quad .$$

The initial amount of potential toxin P_o is then,

$$P_o = C_o - C_o e^{-D/D_o} \quad . \tag{A.6}$$

The solution to Eq (A.2) is,

$$P(t) = P_o e^{-\alpha t} \quad . \tag{A.7}$$

For the active toxin, Eq.(A.3) is,

$$\frac{dA}{dt} + \beta A = \alpha P \quad ,$$

and,
$$\frac{d}{dt}\left(Ae^{\beta t}\right) = \alpha P_o e^{-\alpha t} \cdot e^{\beta t} \quad ,$$

where $e^{\beta t}$ is the integrating factor.

Integrating,
$$\int_{A_o}^{Ae^{\beta t}} d\left(Ae^{\beta t}\right) = \alpha P_o \int_o^t e^{(\beta-\alpha)t} dt \quad ,$$

and solving for A(t) gives,

$$A(t) = \frac{\alpha P_o}{\beta-\alpha} \left(e^{-\alpha t} - e^{-\beta t}\right) \quad , \tag{A.8}$$

where $A_o = 0$.

The maximum UG severity level following an acute exposure is obtained by maximizing A(t). That is, for dA/dt = 0, the time that A is a maximum following acute exposure is obtained as,

$$t_{max} = \left(\ell n \frac{\beta}{\alpha}\right) / (\beta-\alpha) \quad . \tag{A.9}$$

Then the maximum UG severity is,

$$S_{max} = 1 + 4 \left\{1 - \exp\left[-\left[A\left(t_{max}\right) / A_{0.5}\right]^{\gamma}\right]\right\} \tag{A.10}$$

A.2 CONSTANT DOSE RATE EXPOSURE.

The UGIDM differential equations for constant dose rate exposure are,

$$\text{depletable reservoir:} \quad \frac{dC}{dt} = \left(\frac{R}{D_o}\right) C + \mu \left(C_o - C\right) \tag{A.11}$$

$$\text{potential toxin:} \quad \frac{dP}{dt} = \left(\frac{R}{D_o}\right) C - \alpha P \tag{A.12}$$

$$\text{active toxin:} \quad \frac{dA}{dt} = \alpha P - \beta A \tag{A.13}$$

The solution to Eq. (A.11) is given by Eq. (A.5) above. Multiplying by the integrating factor $e^{\alpha t}$, Eq. (A.12) may be given by,

$$\frac{d}{dt}\left(Pe^{\alpha t}\right) = \frac{R_o}{D_o}\left[\frac{\alpha C_o}{K}\left(1 - e^{-Kt}\right) + C_o e^{-Kt}\right] e^{\alpha t} \quad .$$

Integrating,

$$\int_{P_o}^{Pe^{\alpha t}} d\left(Pe^{\alpha t}\right) = \frac{R}{D_o}\int_o^t \left[\frac{\mu C_o}{K}\left(1 - e^{-Kt}\right) + C_o e^{-Kt}\right] e^{\alpha t} dt \quad ,$$

and solving for P(t) gives,

$$P(t) = \frac{RC_o}{KD_o}\left[\frac{\mu}{\alpha}\left(1 - e^{-\alpha t}\right) - \left(\frac{K-\mu}{\alpha-\mu}\right)\left(e^{-Kt} - e^{-\alpha t}\right)\right] + P_o e^{-\alpha t} \quad . \tag{A.14}$$

Multiplying by the integrating factor $e^{\beta t}$, Eq (A.13) may be given by,

$$\frac{d}{dt}\left(Ae^{\beta t}\right) = \left\{\frac{\alpha RC_o}{KD_o}\left[\frac{\mu}{\alpha}\left(1 - e^{-\alpha t}\right) - \left(\frac{K-\mu}{\alpha-\mu}\right)\left(e^{-Kt} - e^{-\alpha t}\right)\right] + \alpha P_o e^{-\alpha t}\right\} e^{\beta t} \quad .$$

Integrating,

$$\int_{A_o}^{Ae^{\beta t}} \left(Ae^{\beta t}\right) = \alpha P_o \int_o^t e^{(\beta-\alpha)t}\, dt$$

$$+ \frac{RC_o}{KD_o} \int_o^t \left\{\mu\left[e^{\beta t} - e^{(\beta-\mu)t}\right] - \alpha\left(\frac{K-\mu}{\alpha-\mu}\right)\left[e^{(\beta-K)t} - e^{(\beta-\alpha)t}\right]\right\} dt \quad ,$$

and solving for A(t) gives,

$$A(t) = \frac{RC_o}{KD_o}\left[\frac{\mu}{\beta}\left(1 - e^{-\beta t}\right) + \frac{1}{\beta-\alpha}\left(\mu - \frac{K-\mu}{\alpha-\mu}\right)\left(e^{-\beta t} - e^{-\alpha t}\right)\right.$$

$$\left. - \frac{\alpha}{\beta-K}\left\{\left(\frac{K-\mu}{\alpha-\mu}\right)\left(e^{-Kt} - e^{-\beta t}\right)\right\}\right] + \frac{\alpha P_o}{\beta-\alpha}\left(e^{-\alpha t} - e^{-\beta t}\right) + A_o e^{-\beta t} \qquad (A.15)$$

Eq. (A.15) gives the active toxin value as a function of exposure time. When P_o, $A_o \neq 0$, they would be residual values from previous exposure(s) referenced from t=0 at the beginning of the current constant dose rate exposure; otherwise, P_o, $A_o = 0$ and the last two terms are zero. For this condition, the time that A is a maximum during irradiation (which may the time irradiation ceases) can be determined for $dA(t)/dt = 0$; then when P_o, $A_o = 0$, a transcendental relationship results which must be solved for $t = t_{max}$ numerically, i.e., of the form below,

$$e^{-(K-\beta)t} + Ae^{-(\alpha-\beta)t} = B \quad ,$$

where, the A, B, and K are constants that depend on the dose rate R and model parameters.

The active toxin A(t') after irradiation ceases is,

$$A(t') = \frac{\alpha P_o'}{\beta-\alpha}\left(e^{-\alpha t'} - e^{-\beta t'}\right) + A_o'\, e^{-\beta t'} \qquad (A.16)$$

where, t' is the time measured after irradiation ceases, starting from zero at that point, and P'_o and A'_o are the existing values then for P and A; they are given by Eqs. (A.14) and (A.15), respectively, for P_o, $A_o = 0$. The time A(t') is a maximum after irradiation ceases is determined for, dA(t')/dt' = 0, given by,

$$t'_{max} = (\beta-\alpha)^{-1} \ln\left[1 - \frac{A'_o}{P'_o}\left(\frac{\beta-\alpha}{\alpha}\right)\right] \qquad (A.17)$$

The maximum severity level of UG distress is then given by $S(t'_{max})$ according to Eq. (A.10).

APPENDIX B

INCIDENCE OF NAUSEA AND VOMITING WITHIN 24 HOURS IN NUCLEAR ACCIDENT EXPOSURES

A log likelihood analysis was performed based on 40 different cases of accidental acute exposure of humans to nuclear radiation. Five different models including the normal, lognormal, Weibull, logistic, and loglogistic forms were applied to fit nausea and vomiting response data for the first 24-h postexposure. Two sets of calculations were performed based on somewhat different internal whole-body dose estimates for 13 of the 40 cases. Most of the accidents involved mixed neutron and gamma radiation exposure, and an RBE = 1 was assumed for analyzing the nausea and vomiting responses.

ED_{50} estimates range from 148 to 177 cGy for nausea and 160 to 190 cGy for vomiting when all five models and both data sets are considered. All of the models fit the data reasonably well; the lowest χ^2 values were obtained for the lognormal, Weibull, and loglogistic models. Based on data set I and the lognormal model, ED_{50} values of 157 ($^{+58}_{-43}$) cGy were obtained for nausea and 170 ($^{+60}_{-45}$) cGy for vomiting. The ED_{50} values for the alternative data set are essentially the same, differing by about 9 cGy.

Calculations were performed to estimate the incidence of nausea and vomiting for acute radiation exposure in order to establish an acute (or high dose rate) basis relevant to our study of protracted radiation exposure. The population we chose to analyze for this purpose consists of 40 nuclear accident cases documented in the literature where total body radiation exposures were all delivered in the order of minutes or less at rates exceeding ~2000 cGy h^{-1}. Much of the large body of clinical information for total body irradiation (TBI) of patients with various malignant diseases also involves acute radiation exposure rates. However, the prodromal response is altered to varying degrees due to chemotherapy often administered prior to TBI, premedication (antiemetics, sedatives, analgesics, and steroids) as well as the health condition and well-being of the patients.

Accordingly, we avoided the use of clinical TBI data because of these limitations.

B.1 DATA.

Nuclear accident data used for this analysis of the incidence are given in Table 31. The data covering a variety of incidents are adapted from the literature. Each of the 40 cases are for an individual accidentally exposed to acute ionizing radiation and are labeled (column 1) designating geographical location and specific clinical case (see Table 31 footnote); in some instances, more than one individual was exposed during a given incident. Specifically for the regression analysis, we utilized the dose values given in column 4 and the response data in columns 5 and 6 where "X" indicates a positive quantal response.

Most of the case data in Table 31 were originally compiled by Thoma and Wald [1959]. However, based on subsequent dosimetric analysis, some of the originally reported dose values were revised downward to reflect internal dose estimates to the midline of the body or the mean bone marrow dose. Table 31 includes the revised dose values as well as the doses for additional accident cases given in the other references [Lushbaugh, 1969; Hübner and Fry, 1980; Klener et al., 1986; and Fanger and Lushbaugh, 1967] listed in Table 31. More recently, Baverstock and Ash [1983] also performed additional dosimetric analysis of the Oak Ridge (OR) and Yugoslavian (Y) accidents. They estimated somewhat lower dose values for 13 of the 40 accident cases, as given by the values in parenthesis in Table 31. Our analysis considered both sets of dose data.

A large majority (about 80%) of the accident cases shown in Table 31 involve substantially varying proportions of mixed neutron and gamma radiation (column 3) due to nuclear criticality incidents. Data are lacking, however, to assign any neutron RBE effect to the nausea and vomiting response in humans. Accordingly, we have tacitly assumed an RBE of unity for those prodromal responses, and the neutron and gamma doses (in gray units) are utilized additively.

Table 31. Nuclear accident cases--nausea and vomiting within 24 hours.

Case[a]	Reference[b]	Dosimetry		Prodromal Response	
		n/γ Ratio	Internal Dose (cGy)[c]	Nausea	Vomiting
LA2	1,2	80	8.1	--	--
LA10	1,2	3.5	9.0	--	--
A4	1	0.08	10.8	--	--
LA9	1,2	3.0	12.0	--	--
LA8	1,2	3.0	16.0	--	--
OR8(H)	1,2,3	0.36	22.8	--	--
LA7	1,2	3.7	42.0	--	--
A3	1	0.1	60.5	--	--
LA6	1,2	4.6	62.0	--	--
OR6(F)	1,2,3,4	0.36	68.5(66.5)[d]	--	--
OR7(G)	1,2,3,4	0.36	68.5(66.5)	--	--
P(A)	2	0.0	100.0	--	--
A2	3	0.083	125.6	--	--
C	5	0.0	140.0	--	--
Y6(B)	1,2,4,6	0.28	145.0(127.0)	X	--
A1	1	0.098	159.2	X	X
UT/CARL	2	0.0	165.0[e]	--	X
LA4	1,2	6.4	192.0	X	X
NJ(2)	2	0.0	200.0	X	X
Y5(H)	1,2,4,6	0.26	226.0(201.0)	X	X
OR5(E)	1,2,3,4	0.36	236.0(225.0)	X	--
OR4(B)	1,2,3,4	0.36	270.0(265.0)	X	X
Y4(G)	1,2,4,6	0.28	290.0(216.0)	X	X
Y2(D)	1,2,4,6	0.28	293.0(217.0)	X	X
Y3(M)	1,2,4,6	0.27	298.0(267.0)	X	X
R2	1	?	300.0	X	X
P(B)	2	0.0	300.0	X	X
Y1(V)	1,2,4,6	0.26	305.0(273.0)	X	X
LA1	1,2	0.55	310.0	X	X
OR3(D)	1,2,3,4	0.36	327.0(315.0)	X	X
OR2(C)	1,2,3,4	0.36	339.0(330.0)	--	--
OR1(A)	1,2,3,4	0.36	365.0(350.0)	X	X
NJ (1)	2	0.0	410.0	X	X
R1	1	?	450.0	X	X
B	2	0.1	550.0	X	X
P(C)	2	0.0	600.0	X	X
LA3	1,2	8.8	1114.0	X	X
I	2	0.0	1200.0	X	X
LA11(K)	7	0.25	4500.0	X	X
RI(P)	7	0.33	8800.0	X	X

[a]Case nomenclature relates to that reported in the literature; numbers and/or letters that may be parenthetical following the geographical location keys given below, designate specific individuals.

LA: Los Alamos A: Argonne RI: Rhode Island P: Pittsburgh
NJ: New Jersey R: Russia UT: U. of Tennessee OR: Oak Ridge
B: Belgium I: Italy C: Czechoslovakia Y: Yugoslavia

[b]Key to References:

1. Thoma and Wald, 1959.
2. Hübner and Fry, 1980.
3. Andrews et al., 1959.
4. Braverstock and Ash, 1983.
5. Klener et al., 1986.
6. Lushbaugh, 1969.
7. Fanger and Lushbaugh, 1967.

[c]Midline body or mean bone marrow dose neutron (RBE = 1).

[d]Dose estimates in parenthesis from Braverstock and Ash, 1983.

[e]Average dose to stomach and intestines.

B.2 ANALYSIS.

The data in Table 31 were assumed to result from a cumulative distribution, $\theta(D; \alpha,\beta)$, that represents the fraction of individuals with symptoms or signs after an acute dose, D. The two adjustable parameters, α and β, were determined by the maximum likelihood technique [Cox, 1983], which uses the data in binary form. We were then able to avoid the disadvantages of having to divide the data into arbitrary groups, as required for a standard regression analysis.

For each of the 40 data points from Table 31, the fractional incidence, y_i, for the ith point is unity if the symptom occurred (X), or zero if not (--). If the corresponding dose is D_i, then the likelihood function has a factor $\theta(D_i)$ whenever $y_i = 1$, and a factor of $1 - \theta(D_i)$ otherwise, i.e.,

$$L = \prod \theta(D_i)^{y_i} \cdot (1 - \theta(D_i))^{(1 - y_i)} , \qquad (B.1)$$

where the product extends over the 40 points. The best fit to the distribution $\theta(D)$ then results from minimizing the negative log of this function, i.e., we find the parameters of the distribution models which minimize

$$-\ln L = -\sum (y_i \ln\theta(D_i) + (1 - y_i) \ln(1 - \theta(D_i))) . \qquad (B.2)$$

Values for the two adjustable parameters, α and β, were determined for each of the five distributions considered, using a simplex algorithm [Press et al., 1986]. Table 32 gives the values for the α and β parameters. The log likelihood function was then used to develop and calculate the variance-covariance matrix for the optimized values of α and β. The resulting parameter standard deviations (σ_α and σ_β) and covariances ($\mathrm{cov}(\alpha, \beta)$) are also included in Table 32, as are the functional forms for statistical models, $\theta(D; \alpha, \beta)$.

Table 32. Regression Models and Parameters

	Model[a]	α	σ_α	β	σ_β	$cov(\alpha,\beta)$
	Normal	-2.196	0.6798	0.01243	0.003220	-0.001946
Set I	Log-normal	-11.944	4.0604	2.362	0.7654	-3.097
Nausea	Weibull	-11.369	3.6275	2.158	0.6584	-2.380
	Logistic	-4.055	1.4077	0.02391	0.007621	-0.009753
	Log-logit	-22.473	8.6452	4.452	1.661	-14.32
	Normal	-2.289	0.7089	0.01207	0.003206	-0.002053
Set I	Log-normal	-12.024	4.1769	2.342	0.7788	-3.243
Vomiting	Weibull	-11.989	3.9672	2.243	0.7166	-2.835
	Logistic	-4.013	1.3610	0.02180	0.006751	-0.008375
	Log-logit	-21.596	8.1033	4.214	1.534	-12.40
	Normal	-2.097	0.6575	0.01258	0.003326	-0.001946
Set II	Log-normal	-11.363	3.8822	2.274	0.7407	-2.865
Nausea	Weibull	-10.671	3.4010	2.048	0.6256	-2.120
	Logistic	-3.988	1.4194	0.02487	0.008059	-0.01051
	Log-logit	-21.352	8.2970	4.276	1.607	-13.30
	Normal	-2.236	0.7013	0.01248	0.00339	-0.002153
Set II	Log-normal	-12.050	4.2532	2.369	0.8016	-3.400
Vomiting	Weibull	-11.498	3.7530	2.175	0.6864	-2.568
	Logistic	-4.071	1.4277	0.02359	0.007621	-0.01006
	Log-logit	-22.210	8.6266	4.374	1.651	-14.21

[a]Models:

Normal	$\theta(D) = \Phi(\alpha + \beta D)$
Log-normal	$\theta(\ln D) = \Phi(\alpha + \beta \ln D)$
Weibull	$\theta(\ln D) = 1 - \exp\{-e^{\alpha + \beta \ln D}\}$
Logistic	$\theta(D) = 1/\{1 + \exp[-(\alpha + \beta D)]\}$
Log-logistic	$\theta(\ln D) = 1/\{1 + \exp[-(\alpha + \beta \ln D)]\}$

B.3 RESULTS.

Using the regression relationship, dose response calculations were performed based on the dual data sets (data sets I and II). Data set II differs from set I only for the dose values in parentheses in Table 31, reflecting the estimates of Baverstock and Ash [1983]. Dose response probability for nausea and vomiting are expressed in Tables 33 and 34.

Table 33 summarizes the effective 10, 50, and 90 percentile doses based on data set I for the incidence of nausea and vomiting for each of the five statistical models. Also included are the 90 percent confidence limits for each value. Table 34 gives analogous information for data set II. Based on the χ^2 goodness-of-fit statistic, the normal and logistic distribution models seem to provide the least best fits of the data which is not surprising since those models do not predict zero incidence at zero dose. Actually, all the models fit both sets of data reasonably well, and which one that is chosen to represent the response becomes a matter of preference.

Nausea and vomiting response curves are plotted in Figs. 92 and 93 based on the analysis of data set I using the log-normal distribution model (graphic results for data set II are not plotted due to the similarity with data set I). Doses in Fig. 92 are midline tissue (MLT) absorbed dose values, and in Fig. 93 they are free-in-air (FIA) exposure values. For convenience, dual plots are shown: the top row are plots of incidence with linear probability (vertical) scales, while the bottom row has nonlinear (probability) scales that produce straight line plots for incidence; in both cases, the abscissas are log dose. The 90 percent confidence bounds (dashed lines) are also indicated in the plots.

The question of neutron RBE still remains an open one, and it is unlikely that it will be resolved empirically for humans short of possibly future clinical experience utilizing neutron radiation therapy. With regard to laboratory work with monkeys, Young [1986] has suggested that neutrons are more effective than gamma radiation in producing emesis. On the other hand, based on reactor irradiation studies with dogs, Cordts et al. [1985] found neutrons less effective

Table 33. Effective Doses (ED) and 95% Confidence Intervals for 10, 50, and 90 percent Nausea and Vomiting Probabilities Within 24 Hours Postexposure Basis: Data Set I (N = 40, ν = 38 dgf.)

Model	Nausea ED_{10}	Nausea ED_{50}	Nausea ED_{90}	χ^2	α
Normal	73.6 ± 76	177 ± 49	280 ± 68	53.3	0.051
Log-normal	91.3^{+69}_{-39}	157^{+58}_{-43}	270^{+111}_{-79}	34.7	0.62
Weibull	68.4^{+84}_{-38}	164^{+70}_{-49}	285^{+94}_{-71}	35.1	0.61
Logistic	77.7 ± 74	170 ± 48	261 ± 76	64.9	0.005
Log-logistic	95.0^{+68}_{-40}	156^{+53}_{-40}	255^{+117}_{-80}	38.5	0.45

Model	Vomiting ED_{10}	Vomiting ED_{50}	Vomiting ED_{90}	χ^2	α
Normal	83.5 ± 78	190 ± 50	296 ± 70	37.7	0.48
Log-normal	98.3^{+75}_{-42}	170^{+60}_{-45}	294^{+122}_{-86}	27.9	0.89
Weibull	76.8^{+91}_{-42}	178^{+70}_{-50}	304^{+100}_{-75}	28.3	0.87
Logistic	83.3 ± 79	184 ± 50	285 ± 79	40.1	0.38
Log-logistic	99.8^{+73}_{-42}	168^{+57}_{-43}	283^{+133}_{-91}	29.4	0.85

Table 34. Effective Doses (ED) and 95% Confidence Intervals for 10, 50, and 90 percent Nausea and Vomiting Probabilities Within 24 Hours Postexposure Basis: Data Set II (N = 40, ν = 38 dgf.)

Model	Nausea ED_{10}	ED_{50}	ED_{90}	χ^2	α
Normal	64.5 ± 74	166 ± 47	268 ± 67	59.0	0.02
Log-normal	84.2^{+66}_{-37}	148^{+55}_{-40}	260^{+111}_{-78}	36.4	0.54
Weibull	61.1^{+79}_{-34}	153^{+67}_{-46}	275^{+97}_{-72}	36.1	0.56
Logistic	72.0 ± 72	160 ± 44	249 ± 40	76.4	<0.001
Log-logistic	88.2^{+67}_{-38}	148^{+51}_{-38}	247^{+114}_{-78}	39.0	0.43

Model	Vomiting ED_{10}	ED_{50}	ED_{90}	χ^2	α
Normal	76.4 ± 75	179 ± 47	282 ± 69	43.5	0.25
Log-normal	94.3^{+71}_{-40}	162^{+56}_{-42}	278^{+115}_{-82}	30.7	0.79
Weibull	70.3^{+84}_{-38}	167^{+66}_{-47}	290^{+100}_{-74}	30.3	0.80
Logistic	79.4 ± 75	173 ± 45	266 ± 74	51.7	0.07
Log-logistic	97.1^{+69}_{-40}	160^{+52}_{-39}	265^{+121}_{-83}	33.6	0.67

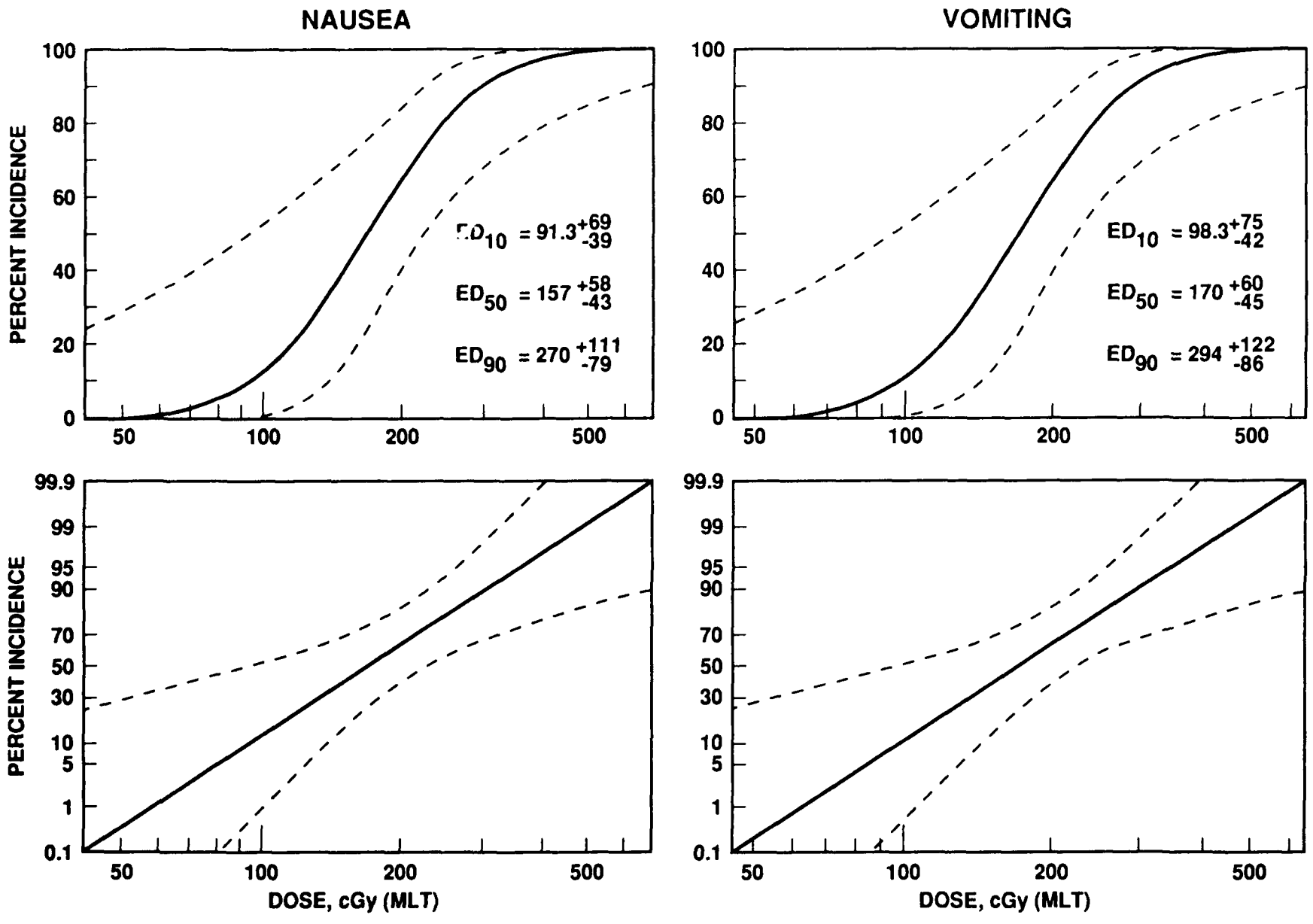


Figure 92. Incidence of nausea and vomiting within 24 hours (data set I, lognormal model).

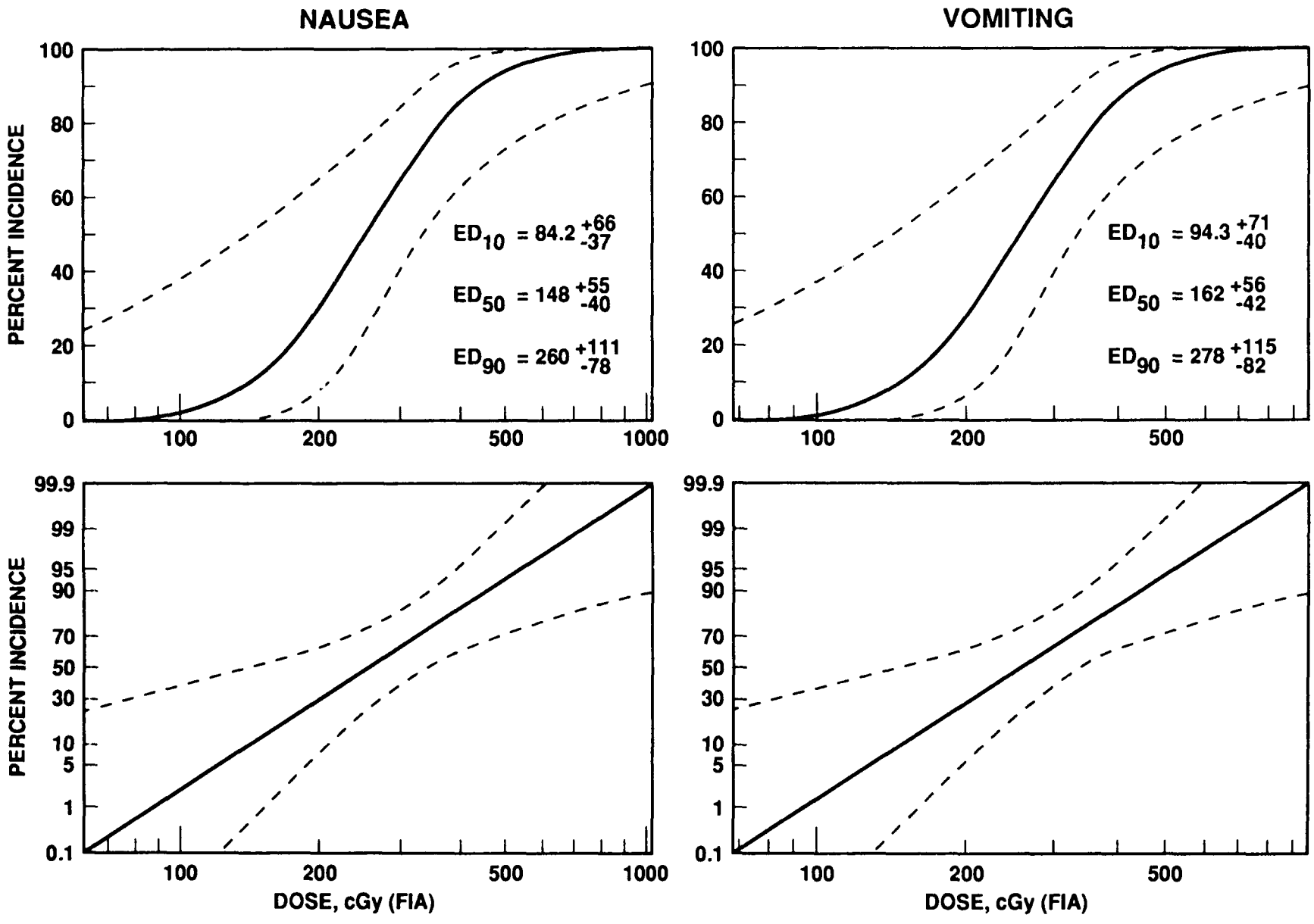


Figure 93. Incidence of nausea and vomiting within 24 hours (data set I, lognormal model).

(RBE = 0.48) in producing emesis. Before a more definitive application of RBE can be specified for emesis in humans, further animal experimentation will be required together with a more thorough delineation of the radiation-induced mechanisms.

As an exercise to illustrate the effect of neutron RBE, we performed some additional likelihood calculations utilizing a lognormal model to analyze the accident data (data set I). Based on the n/γ ratios given in column 3 of Table 31, we assumed various values for the neutron RBE ranging from 0.5 to 2.0 and reanalyzed the vomiting response for those conditions. The calculated neutron RBE effect obtained is shown in Fig. 94 for the ED_{50} emesis endpoint both in terms of MLT and FIA dose; it amounts to about a 33 percent increase in the ED_{50} for a factor of four decrease in RBE.

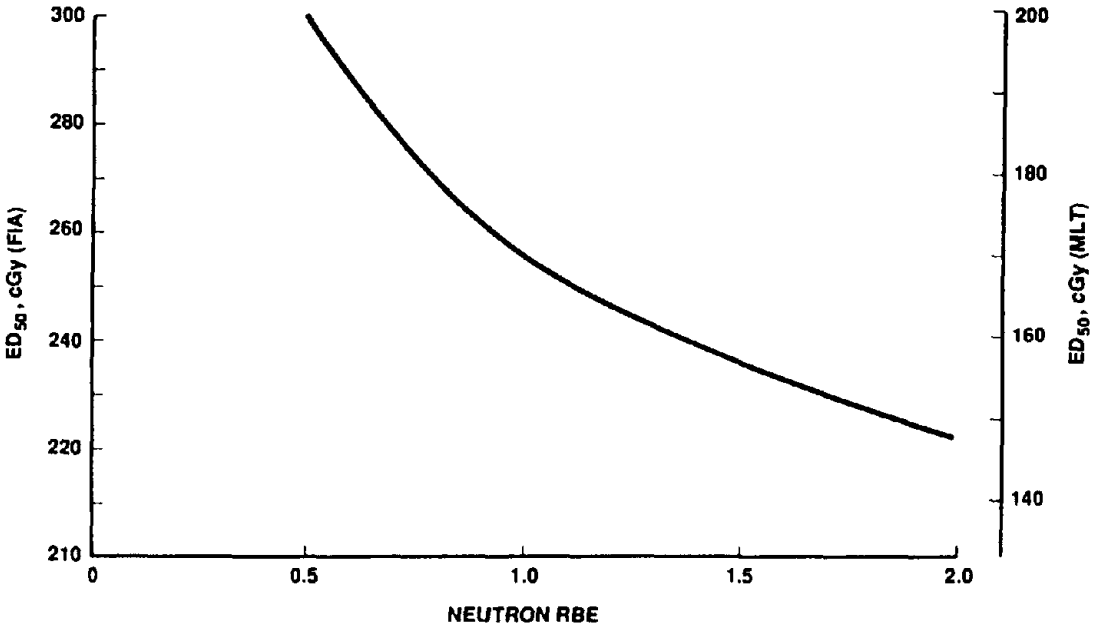


Figure 94. ED_{50} for emesis versus neutron RBE (basis: data set I; lognormal model).

APPENDIX C

EQUATIONS OF THE GUT INJURY MODEL

This appendix presents the equations for the gut injury model (GIM). The physiological and anatomical basis for the model and the structure of the model are discussed in Sec. 5 of this report.

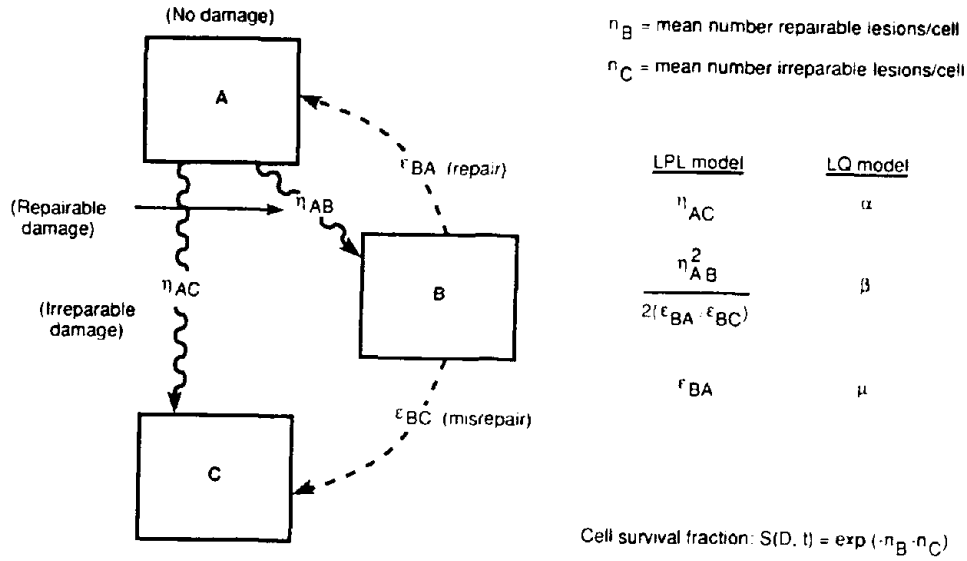
The foundation for the radiation response of the GIM is the lethal potentially lethal (LPL) model. Curtis [1986] has published a full discussion of the LPL model and its application to a wide range of radiobiological response data. That model includes two types of radiation-induced chromosome lesions, lethal lesions, and potentially lethal lesions. The lethal lesions, having an average number of n_C per cell, are irreparable and prevent mitosis. A cell may have more than one lethal lesion, but any one is sufficient to cause reproductive death of the cell. The potentially lethal (PL) lesions, having an average number of n_B per cell, are repairable by a process based on linear kinetics. All PL lesions can be repaired in time; however, a cell that enters mitosis with an unrepaired PL lesion will fail to divide and be reproductively dead. The LPL model also includes a misrepair term that generates lethal (irreparable) lesions from PL lesions at a rate that is quadratic in the concentration of PL lesions. This nonlinear term produces a shoulder on the cell survival curve and provides a link to the linear-quadratic (LQ), or alpha/beta, model frequently used to analyze cell survival data.

The equations for the LPL model are:

$$\dot{n}_B = \eta_{AB} R - \epsilon_{BA}\, n_B - \epsilon_{BC}\, n_B^2 \quad , \tag{1}$$

$$\dot{n}_C = \eta_{AC} R + \epsilon_{BC}\, n_B^2 \quad , \tag{2}$$

where R is the dose rate. Both types of lesions are produced at a rate proportional to R. Figure 95 provides a diagrammatic definition of the rate coefficients and variables in these equations and shows



Source: Thames (1985), based on Tobias et.al. (1980), and Curtis (1982).

Figure 95. Cell radiation response based on the lethal, potentially lethal (LPL) cell lesions model.

the correspondence with the parameters customarily used in the LQ model.

In the LPL model, the distribution of each type of lesion among a group of cells is assumed to follow a Poisson distribution since the lesions are produced at random by radiation exposure. This assumption leads to the exponential expression for cell survival shown in Fig. 95. The expression is simply the probability that a given cell has no lesions of either kind at the time it enters mitosis.

We had to address a major problem crucial to applying the LPL model to a proliferating tissue. Previously, the LPL model had been applied only to data where the exposure and repair times were short compared to the cell cycle. The difficulty for longer times arises from the fact that proliferation disrupts the Poisson distribution of lesions. Successful cell division occurs in cells with no lesions, lowering the average number of lesions per cell but not in a random manner. Likewise, the death of a cell entering into mitosis due to the presence of one or more lesions removes any or all lesions present. We therefore had to develop equations that would track both lesion number and cell number over long periods of time given any

exposure history. In addition, we needed to replace the simple cell survival formula shown in Fig. 95 with a formulation based on these same principles.

Probably the most straightforward analytic approach to this problem would be a Monte Carlo procedure for tracking a large number of cells. The distribution of lesions would then evolve into a non-Poisson form as it must do so in reality. To avoid the extensive calculation requirements of a Monte Carlo model, we derived an approximate solution based on continuous time differential equations. We refer to our formulation as the proliferation and intracellular repair (PAIR) model.

The key to this approximate solution was to divide the proliferating cells into three classes, labeled A, B, and C, in a manner similar to Curtis's classes of lesions. However, it is important to point out that in the PAIR model, the classes refer to cells and not to lesions. The number of cells in each class and the definitions of the classes are as follows:

N_A = number of cells with no lesions of either kind (uninjured cells),

N_B = number of cells with one or more PL lesions, but no lethal lesion (injured cells), and

N_C = number of cells with at least one lethal lesion (mitotically dead cells).

Proliferation increases the number of cells in class A in the normal manner but does not occur in class C since those cells are mitotically dead. Proliferation (attempted division) of class B results in cell death and moves them to class C since we assume, following Curtis, that any cell entering mitosis with a PL lesion suffers mitotic death.

It is not necessary to track the number of lesions in class C cells since they are mitotically dead. However, we track the number of C cells because they are still present in the tissue. We assume that their presence affects homeostasis and that they differentiate to form mature, functional cells. These assumptions seem to fit the

dynamics of the intestinal epithelium but may not be appropriate for every type of tissue.

The only lesion number that must be tracked in the PAIR model is that of the class B cells, where:

n_B = mean number of PL lesions in class B cells.

Note that by the definition of class B cells, n_B is always greater than or equal to one. Therefore, n_B is not the average number of PL lesions as defined in the LPL model since that number goes to zero in the absence of radiation exposure. For convenience, we define a different number that does correspond to the LPL model:

n_{PL} = mean number of PL lesions in a hypothetical pool of cells with a Poisson distribution of PL lesions and for which the number of cells with one or more PL lesions and no lethal lesions is N_B.

In other words, we approximate the actual distribution of lesions in the B class of cells by assuming that it is a truncated Poisson distribution from this hypothetical pool of cells. The accuracy of this assumption remains to be investigated. The advantage of the assumption is that proliferation of class A cells can occur without upsetting the statistical distribution of lesions in class B cells. Furthermore, in the limit of no proliferation, the mathematical results match those of the LPL model.

The value of n_B in terms of n_{PL} is given by:

$$n_B = \frac{n_{PL}}{1 - e^{-n_{PL}}} \tag{3}$$

The rate equations for the average number of PL lesions per cell in the injured compartment is:

$$\dot{n}_{PL} = \eta_{AB} RE \quad - \quad \left(n_B - 1\right)\left(\frac{N_A}{N_B}\right)\eta_{AB} RE$$

Exposure Dilution

$$- \epsilon_{BA} n_{PL} - \epsilon_{BC} n_{PL} n_B \left[2 - \left(n_B - n_{PL}\right)\right] E \quad , \tag{4}$$

Repair Misrepair

where R is the radiation exposure rate (Gy/h),

$$E = \left(\frac{n_{PL}}{n_B}\right) \quad \frac{1}{1 - \left(n_B - n_{PL}\right)} \quad ,$$

$$\text{and } \lim_{\substack{n_{PL} \to 0 \\ n_B \to 1}} E = \lim \frac{1}{n_B(n_B - 1/2)} = 2 \quad . \tag{5}$$

The rate equation for the number of uninjured cells N_A is:

$$\dot{N}_A = \lambda N_A - \left(\eta_{AB} + \eta_{AC}\right) R N_A$$

Proliferation Exposure Losses

$$+ \epsilon_{BA} \left(n_B - n_{PL}\right) N_B - \theta N_A \tag{6}$$

Repair of Injured Cells To Pipeline

The coefficient θ which determines the rate at which clonogens feed the "pipeline" to the transit compartment is defined below, as is the clonogen division rate λ.

The rate equation for the number of injured cells N_B (potentially lethal injury only) is:

$$\dot{N}_B = -\lambda N_B + \eta_{AB} R N_A - \eta_{AC} R N_B$$

Mitotic Death | Exposure of Uninjured Cells | Exposure Causing Lethal Injury

$$- \epsilon_{BA}\left(n_B - n_{PL}\right) N_B - \epsilon_{BC} n_{PL} n_B N_B - \theta N_B \quad (7)$$

Repair | Misrepair | Pipeline

The rate equation for the number of killed cells N_C (lethally injured or mitotically dead, but still functional) is:

$$\dot{N}_C = \eta_{AC} R\left(N_A + N_B\right) + \epsilon_{BC} n_{PL} n_B N_B + \lambda N_B - \theta N_C \quad (8)$$

Exposure | Misrepair | Mitotic Death of Injured Cells | Pipeline

The instantaneous clonogen division rate λ is affected by both homeostasis and mitotic delay as given by:

$$\lambda = HM\gamma \quad (9)$$

where,

H = homeostasis factor(dimensionless)

M = mitotic delay factor (dimensionless)

γ = normal division rate to balance attrition (h^{-1}).

Normal conditions give H = M = 1 and $\lambda = \gamma$. The maximum value of λ is λ_m, determined by H. The minimum value of λ is 0, determined by M.

Radiation exposure slows down cell division through a mitotic delay process. For the GIM, we have modeled this process based on a saturable enzyme repair concept where a hypothetical damage level Q is reduced by the action of a finite pool of repair enzymes. Assuming cell cycling proceeds normally when the repair enzymes are not activated, and slows down when they are activated, we employed a

Michaelis-Menten form to describe activated enzyme saturation. This results in our rate equation to express cellular damage given by:

$$\dot{Q} = \underbrace{R}_{\substack{\text{Damage} \\ \text{Production}}} - \underbrace{\left(\frac{\lambda_m}{\delta}\right)\frac{Q}{A+Q}}_{\substack{\text{Damage} \\ \text{Repair}}} \qquad (10)$$

Q = cellular damage (Gy)

λ_m = freely growing (maximum) cell division rate (h^{-1})

δ = acute dose mitotic delay constant (Gy^{-1})

A = threshold for saturation of repair = 0.1 Gy

R = dose rate (Gy/h)

The mitotic delay factor M in Eq. (9) above is then given by:

$$M = [1-\tanh(Q/A)] \qquad (11)$$

When the damage level Q is low, M approaches one and cell division proceeds normally. When the damage level increases beyond the characteristic value A, M rapidly approaches zero according to the hyperbolic tangent function, effectively halting the progression of cell cycling.

An H-tissue such as the intestinal epithelium must have communication mechanisms between compartments in order to maintain homeostatic equilibrium. Figure 81 in Sec. 5 illustrates the control mechanisms that govern the GIM model. Many links are possible, and the nature and number of links have not yet been established experimentally. We have attempted to choose the simplest and minimum number of (feedback) control links that will both maintain equilibrium in the three GIM compartments and provide a correct qualitative response to radiation insult.

The primary homeostasis loop is the control of clonogen proliferation through the division rate λ. When the tissue is in

equilibrium with N_o clonogenic cells, the normal division rate is γ expressed in units of divisions per hour per cell. Compensatory proliferation is accomplished through the homeostatic control factor H. In equilibrium, both factors M and H are equal to 1, and the production rate of new clonogens is N_o, just balancing the attrition rate from the villi to the intestinal lumen.

The mathematical form of the homeostasis factor H is presented in Fig. 96. The primary purpose of H in the GIM is to increase the cell division rate to its maximum value λ_m when the population of the transit compartment drops to zero. At first, we used a linear dependence on N_T, but found that the delayed recognition of damage was not properly represented. This phenomenon shows up in both fractionated [Withers and Elkind, 1969] and constant dose rate [Withers, 1972] studies. In spite of cell depletion, it is found that rapid proliferation is delayed for at least two days. We introduce this behavior by using a small value for the exponent a = 0.21, to moderate the increase in cell division rate as N_T decreases. A final factor $f(N/N_o)$, shown in Fig. 96, is used to limit the growth of the clonogenic compartment to twice its normal value. As explained later, N_T is not allowed to go above its equilibrium value N_{To}, so that the

$$H = 1 + \frac{\lambda_m - \gamma}{\gamma}\left[1 - \left(\frac{N_T}{N_{T0}}\right)^a\right] f\left(\frac{N}{N_0}\right)$$

N_T equilibrium — Limit on N

f = population limiting factor

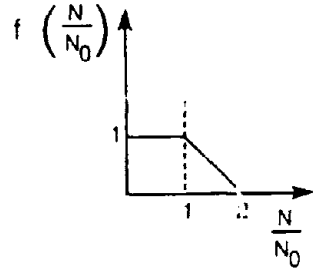


Figure 96. Clonogen homeostasis factor, H.

factor H will not drop below 1. This restriction ensures a supply of cells to balance the normal attrition rate unless overridden by mitotic delay.

The GIM model uses the transit population N_T rather than the villi population N_V to control compensatory proliferation mainly on the grounds of physical proximity. We do not have experimental data to support this choice. In fact, it might be useful to include N_V in the equation for H as an attempt to improve the description of delayed recognition of damage.

The remaining control mechanisms shown in Fig. 81 of Sec. 5 involve the fluxes of cells in and out of the three compartments. Figure 97 shows the mathematical expressions for the fluxes. The attrition rate from the villus has the simplest mathematical form. It is held fixed at the normal level γN_0 when the villus population is between 0 and its equilibrium value N_{V0}. When the population is zero, the villus output is set equal to its input if the input is less than γN_0 and is equal to γN_0 otherwise. The assumption is that the villus has atrophied completely and will not grow until the supply of cells from the transit compartment exceeds the normal attrition rate. When

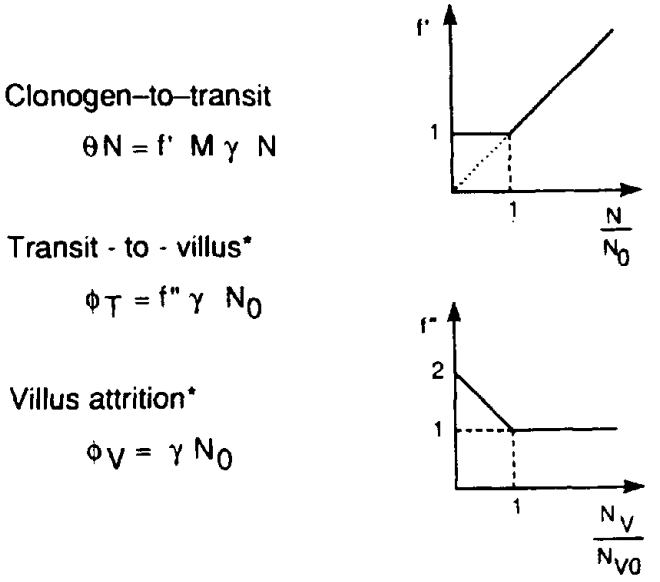


Figure 97. Cell fluxes between compartments in the GIM model; note that ϕ is subscripted, but θ is not in the equations.

the population reaches its full value N_{Vo}, any input level that exceeds γN_o is passed on through in order to limit the villi to their normal size.

The flux of cells from the transit compartment to the villus is increased linearly from its normal value γN_o to twice that value as the villus level drops from normal to zero. Like the villus, the transit compartment is assumed to shrink to zero size when it is empty so that output is equal to the input if the input is less than the output demanded by the villus based on the function $f''(N_V/N_{Vo})$. Also, when the transit compartment reaches full size, it passes excessive inputs on through in order to limit its size to N_{To}.

The output from the clonogen compartment is more complex. Like the others, it is designed to provide a flux equal to the attrition rate γN_o at equilibrium. The flux θN shown in Fig. 81 of Sec. 5 is defined in Fig. 97. The mitotic delay factor M appears in the output flux θN as well as in the division rate λ. The assumption is that if mitotic delay turns off cell division, it will also turn off cell differentiation that moves cells from the clonogen compartment to the transit compartment. Otherwise, mitotic delay in the model causes an anomalous depletion of the clonogen compartment that is not consistent with experimental data.